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SURGERY

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SURGERY

VOL. 33 FEBRUARY, 1953 No. 2

Original Communications

Society for Vascular Surgery

THE ANATOMY OF PULMONARY STENOSIS AND ATRESIA WITH COMMENTS ON SURGICAL THERAPY

THOMAS N. P. JOHNS, M.D., G. RAINEY WILLIAMS, M.D., AND ALFRED BLALOCK, M.D., BALTIMORE, MD.

(From the Department of Surgery of The Johns Hopkins University and Hospital)

IN THE tetralogy of Fallot certain anatomic variations occur. The malformation was recognized by Stensen in 1672,[14] Sandifort in 1777,[1] John Hunter in 1783,[10] and Hope in 1830,[9] but the variable patterns of the anomaly were first described in the writings of Peacock in 1866,[12] of Fallot in 1888,[7] and of Keith in 1909.[11]

These variations primarily are seen in the extent and the site of the obstruction to the flow of blood to the lungs through the outlet of the right ventricle. The pulmonary obstruction may be complete (atresia) or partial (stenosis); it may be located at the valve, more frequently at the infundibulum, and occasionally it occurs at both the valve and the infundibulum. Equally variable is the degree of dextroposition of the aorta; this vessel may be only slightly deviated to the right, or it may be dextroposed so greatly as to appear to arise almost entirely from the right ventricle. The remaining two components of the tetralogy, namely, the interventricular communication and the hypertrophy of the right ventricular wall, appear to be less variable features of the anomaly.

Although Thomas Peacock[12] did not differentiate the tetralogy from pulmonary stenosis in which there is no interventricular communication, he was the first to appreciate variations in the anatomy of the pulmonary obstruction. In the classic papers of Étienne Louis-Arthur Fallot[7] on the *Maladie Bleue,* all types of pulmonary stenosis are clearly described, including a case in which there was stenosis of both the infundibulum and the valve. Fallot was also the first to study the nature of the dextroposition of the aorta; he described in detail instances of varying degrees of deviation of the aorta to the right.

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Presidential Address, sixth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 8, 1952.

In 1909 Sir Arthur Keith[11] recognized the following variations in malformations of the pulmonary conus: (1) subdivision of the right ventricle into two chambers by the stenosis, examples of what we have called infundibular stenosis with a well-developed infundibular chamber, (2) arrested expansion of the infundibulum, (3) almost complete arrest of development of the infundibulum, and (4) stenosis due mainly to fusion of the valves. The double stenosis known to Fallot[7] was not described by Keith nor did he note variations in the position of the aorta.

In 1949 Brock[2-5] made careful morphologic observations of pulmonary stenosis. These studies led him to carry out a direct surgical attack upon the obstruction, first in cases of isolated valvular pulmonary stenosis without an interventricular defect, later in instances of infundibular stenosis in the tetralogy of Fallot. Brock emphasized (1) the role of the crista supraventricularis in the formation of the infundibular stenosis, (2) the localized nature of the stenosis in some cases of the tetralogy of Fallot, and (3) the nature of the infundibular chamber. He divided infundibular stenosis into five types depending upon the degree of development of the infundibular chamber.

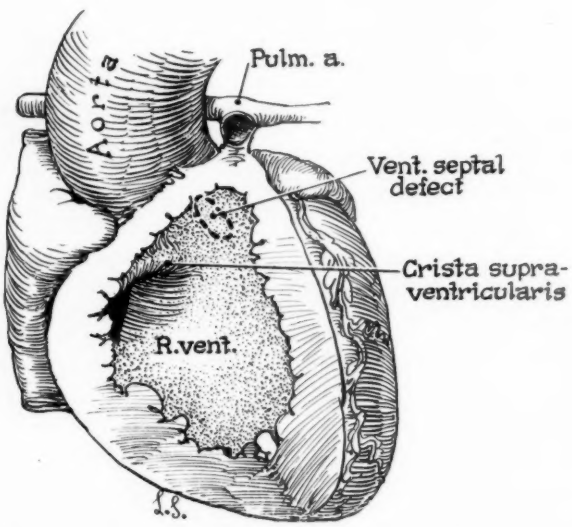

Fig. 1.—Drawing showing size of aorta and degree of dextroposition in a typical specimen of tetralogy of Fallot with pulmonary atresia located at the pulmonary valve. The pulmonary artery is hypoplastic. Details of the atrioventricular valves have been omitted.

RESULTS

We are reporting the results of a study of 95 autopsy specimens of pulmonary stenosis or atresia associated with an interventricular septal defect (tetralogy of Fallot); valvular pulmonic stenosis with an intact ventricular septum is not included. Since 79 of these 95 specimens were obtained from patients who died at varying times following operation, it is permissible to assume that most of them had severe pulmonary stenosis or atresia or complicating conditions such as unusual dextroposition of the aorta.

The 95 specimens studied have been divided into four groups. This classification is based upon the anatomy of the region of the pulmonary conus and valve:

(1) Pulmonary atresia
(2) Infundibular pulmonary stenosis
(3) Valvular pulmonary stenosis
(4) Combined infundibular and valvular pulmonary stenosis.

The second group (infundibular stenosis) has been subdivided according to the size of the infundibular chamber. Table I gives the relative incidence in the several groups in this series.

TABLE I. TETRALOGY OF FALLOT

Total number of cases	95
Pulmonary atresia	22
Pulmonary infundibular stenosis	49
Pulmonary valvular stenosis	10
Pulmonary infundibular and valvular stenosis	14

1. *Pulmonary Atresia.*—In 22 cases of tetralogy of Fallot there was no direct communication between the right ventricle and the pulmonary artery. In 19 cases the atretic area was at the very apex of the conus in the region of the pulmonary valve; in the remaining 3 the atresia was in the infundibulum and a valve was demonstrable (Fig. 1). In 19 of the 22 cases the pulmonary artery was small, in 1 case it was absent, and in the remaining 2 cases it was essentially normal in size. The aorta was larger than normal in most cases, and usually greatly transposed to the right. The enlargement and dextroposition of the aorta appeared to be inversely proportional to the size of the pulmonary artery (Table II).

TABLE II. TETRALOGY OF FALLOT WITH PULMONARY ATRESIA

Total number of cases	22
Development of pulmonary artery	
Normal	2
Small	19
Absent	1
Apparent dextroposition of aorta	
60% or more from right ventricle	18
Less than 60% from right ventricle	4

In one of these cases the pulmonary atresia consisted of a thin diaphragm in the region of the valve which could possibly have been pierced by a sharp instrument if a direct attack had been attempted. It is doubtful, however, whether the exact nature of the deformity could have been recognized at the time of exploration.

2. *Infundibular Pulmonary Stenosis.*—The proximal end of the conus arteriosus was the site of the obstruction in 49 cases. This type of stenosis is produced by a muscular structure which normally separates the inflow tract from the outflow tract of the right ventricle and which has been termed the crista supraventricularis or infundibular crest. This band of cardiac muscle

extends across the superior aspect of the right ventricle from its anteromedian wall to the interventricular septum where it is inserted at the anterior margin of the interventricular communication. The crista supraventricularis hangs down in front of the interventricular defect like an oblique curtain, its inferior border arched to provide an opening into the pulmonary outflow tract. As its projections merge into the ventricular walls, the crista presents two features of practical anatomic significance: It is closely apposed posteriorly to the base of the aortic valve, and its anterior insertion is close to the anteromedian papillary muscle which contains the right ventricular branch of the bundle of His.

The development of the crista supraventricularis determines the type of stenosis at the infundibulum, and its position in the ventricular cavity is responsible for the size of the infundibular chamber. From the viewpoint of the development of the infundibular chamber, the 49 cases in our autopsy series showed a well-developed chamber in 18, moderately well-developed in 13, and poorly developed in 18 cases.

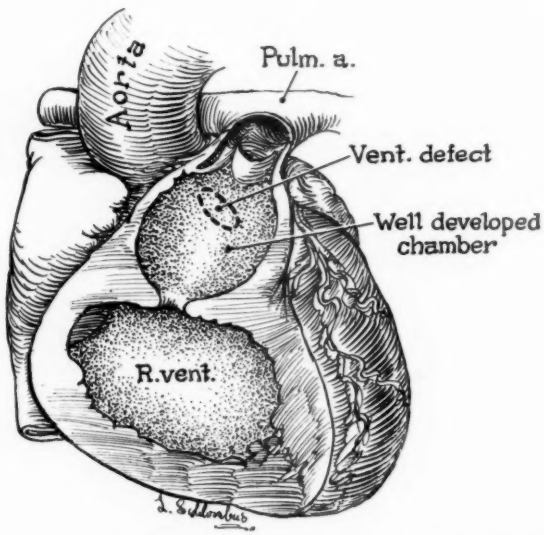

Fig. 2.—Drawing of heart with tetralogy of Fallot with infundibular stenosis and a well-developed infundibular chamber. Expanded chamber above localized stenosis is shown. The pulmonary artery is normal in size.

Well-developed infundibular chamber (Fig. 2): Most of the patients from whom these 18 specimens were obtained fall in an older age group than those in whom there was a high and small infundibular chamber. When the columns of the crista supraventricularis converge to form a muscular diaphragm, the stenosis lies fairly low in the right ventricular cavity. The distance from the crest to the pulmonary valve is relatively great, measuring 18 mm. or more, and the infundibular chamber has a diameter of 3 cm. or more.* In its extreme form the well-developed chamber is known as the third ventricle. This is the

*These measurements were made on fixed specimens and are used only as a method of comparison. Furthermore, though the infundibular chamber does not seem to vary in size in different age groups, an occasional chamber in a very small heart was termed well developed though its measurements fell slightly below those mentioned here.

most favorable situation among the infundibular stenoses for direct surgical attack by the Brock technique.

It is difficult in a fixed specimen, and even in a fresh one, to determine accurately the degree of dextroposition of the aorta. Our observations do indicate fairly clearly that patients with a well-developed infundibular chamber do not usually have as much dextroposition of the aorta as do those with a high small infundibular chamber (Table III). Furthermore, the aortic orifice is usually fairly normal in size in those patients with a well-developed infundibular chamber, whereas it is often much enlarged in those with a small chamber. The size of the pulmonary artery usually varies inversely with that of the aorta.

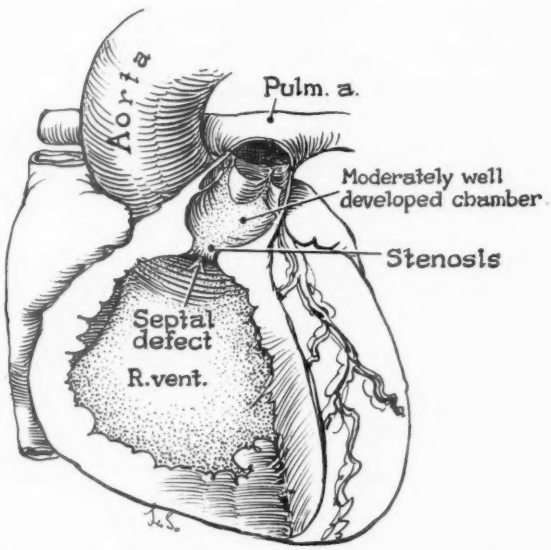

Fig. 3.—Drawing showing tetralogy of Fallot with infundibular stenosis and a moderately well-developed chamber. Note that the crista supraventricularis is higher than in the previous specimen. Its position between the pumonary artery and aortic outflow tracts is demonstrated.

Moderately well-developed infundibular chamber (Fig. 3): In the cases of this type the crista supraventricularis is placed higher in the ventricle, so that its distance from the pulmonary valve measures 12 to 18 mm. In this intermediate group the infundibular chamber has a circumference of 15 to 30 mm. The stenotic area does not present the appearance of a perforated diaphragm. It is the shape of a funnel and is rimmed by a wall of cardiac muscle. This rim is formed by a thickened projection of the anterior ventricular wall at its junction with the infundibular crest. There were 13 cases of this type in this series. It is possible that a direct surgical attack could have been carried out successfully in some of these cases; however, the feasibility of infundibular resection is limited by the diminished size of the infundibular chamber. Resection of this high-lying infundibular curtain is also fraught with the danger of injury to the aortic valve.

Poorly developed infundibular chamber (Fig. 4): A frequent type of infundibular stenosis is that associated with a hypoplastic outflow tract and a

poorly developed infundibular chamber. In 18 of our 49 cases the crista supraventricularis was placed so high in the right ventricular cavity that the stenosis lay less than 12 mm. from the valve. In such cases the infundibular chamber is not only short but narrow. The funnel-like outlet is narrowed by thick muscle which gives the outflow tract the appearance of a small tunnel. In most instances there is a slight expansion of the chamber proximal to the pulmonary valve ring. Only with extreme difficulty can a surgical instrument penetrate such a tunnel, and in this type of high infundibular stenosis the possibility of successful infundibular resection is severely limited. In 14 of the 18 cases there was marked dextroposition of the aorta. Furthermore, the aorta was usually larger and the pulmonary artery smaller than normal.

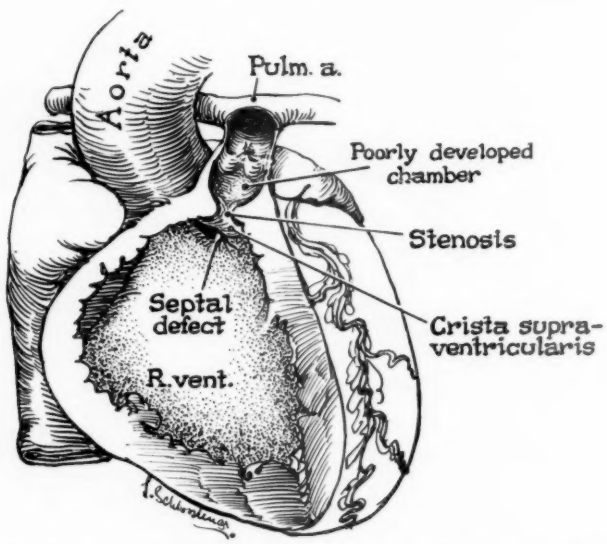

Fig. 4.—Drawing to demonstrate tetralogy of Fallot with infundibular stenosis and a poorly developed chamber. There is usually slight dilatation above the stenosis. The pulmonary valve, as illustrated, is frequently bicuspid. The aorta is large and the pulmonary artery is small.

3. *Valvular Pulmonary Stenosis.*—The stenosis was found to be in the pulmonary valve in 10 cases (Fig. 5). Structurally this type of stenosis is similar to that which occurs in cases of stenosis of this pulmonary valve without a ventricular septal communication. Fallot's[7] description cannot be surpassed:

> The pulmonary artery has the shape of a cone whose apex corresponds to the upper end of the infundibulum; the stenosis at this level is very marked; a transverse section taken two or three centimeters from the base demonstrates a veritable funnel. . . . The valve cusps are raised along the wall of the artery, fused together and form an actual diaphragm, pierced in the center by a narrow opening. The caliber of the aorta on the other hand is very notably enlarged.

The pulmonary artery is generally normal in size or is dilated distal to the stenosis. In some cases there is narrowing of the infundibulum. The aorta straddled the ventricular septum without much deviation to the right in 7 cases. In the remaining 3, 60 per cent or more of its orifice appeared to overlie the right ventricle.

TABLE III. TETRALOGY OF FALLOT; APPARENT DEXTROPOSITION OF AORTA

	TOTAL	60% OR MORE FROM RIGHT VENTRICLE	LESS THAN 60% FROM RIGHT VENTRICLE
Infundibular stenosis	49	27	22
Well-developed chamber	18	7	11
Moderately well-developed chamber	13	6	7
Poorly developed chamber	18	14	4
Pulmonary atresia	22	18	4

4. *Combined Infundibular and Valvular Pulmonary Stenosis.*—In 1888 Fallot described this combination of defects in a 26-year-old man. Stenosis at both the pulmonary valve and the infundibulum occurred in 14 cases in our series (Fig. 6). This combination of defects occurred mainly in patients in the older age group. In approximately one-half of these patients the interposed infundibular chamber was fairly large and a direct attack would have been possible. Such an attack necessitates infundibular resection plus valvulotomy. In 9 of the cases it appeared that 60 per cent or more of the orifice of the aorta was overlying the right ventricle (Table IV).

TABLE IV. TETRALOGY OF FALLOT WITH VALVULAR AND INFUNDIBULAR STENOSIS

Total number of cases	14
Development of infundibular chamber	
Well developed	6
Moderately well developed	2
Poorly developed	6
Dextroposition of aorta	
60% or more from right ventricle	9
Less than 60% from right ventricle	5

DISCUSSION

The primary aim of this paper is the presentation of the morphologic observations, but it may not be amiss to make some comments regarding the surgical therapy of pulmonary stenosis. It is to be stressed that the findings would probably be of greater significance had the tissues been fresh rather than fixed at the time of this study. It is true that we had seen most of the specimens previously when in an unfixed state at the time of autopsy but did not tabulate the different conditions observed. Furthermore, we must remember that these cases may not be representative of the entire group in that fatalities are more likely to occur, with or without operation, in those patients with the most serious malformations. As stated previously, 79 of the 95 specimens were obtained from patients who had been operated upon, the total of these being approximately 1,100. In other words, conclusions in regard to the choice of operative procedure might be different if the exact anatomic deformity in all of the cases was known.

Discussion of the operative procedures will be limited to the choice of (1) an anastomotic procedure in which an artificial ductus is created, or (2) a direct attack in which a stenotic valve or diaphragm is divided or an infun-

dibular resection is performed; details of the type of anastomosis or of the direct attack will not be discussed. It should be understood at this time that the treatment of valvular pulmonary stenosis with an intact ventricular septum should consist of division of the stenotic valve. There is no difference of opinion on this point.

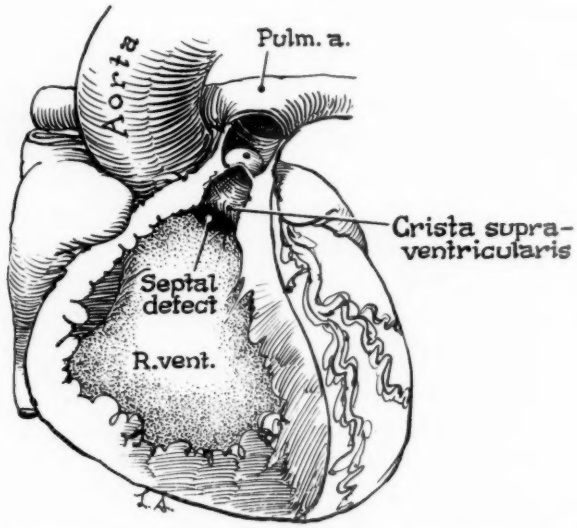

Fig. 5.—Drawing to demonstrate tetralogy of Fallot with valvular pulmonic stenosis. The domelike projection of the fused valve cusps into the pulmonary artery is illustrated. Note that the infundibular area is slightly constricted.

There is considerable difference of opinion on the choice of treatment of the various forms of pulmonary stenosis accompanied by an interventricular defect. In these conditions the aorta receives mixed venous blood from the right side of the heart as well as arterial blood from the left. We shall deal first with those deformities in the treatment of which there seems to be the least difference of opinion. The first of these is pulmonary atresia in which there is no direct communication between the pulmonary artery and the outflow tract. Of our autopsy specimens 23 per cent showed this type of malformation. The treatment consists in an anastomosis between a pulmonary artery, if one is available, and a systemic artery. A successful direct attack is usually an impossibility. A second condition in the treatment of which there is not a great difference of opinion is pulmonary valvular stenosis in association with an interventricular defect; in other words, the tetralogy of Fallot with a valvular rather than an infundibular stenosis. Eleven per cent of our autopsy specimens showed this anomaly. It is our opinion that Brock is correct in advocating that this be treated by a direct attack with division of the pulmonary valve, just as in cases of "pure" valvular stenosis. Brock[5] does qualify his view by stating that the mortality rate associated with division of the valve is high in adults with this deformity and that perhaps in such patients an anastomosis should be performed. Dr. Taussig[13] has been fearful that division of the valve when an interventricular defect is present will convert the condition into an Eisenmenger complex with too great blood flow to the lungs and pulmonary hypertension. We feel con-

fident that this will not happen because there is usually at least a little stenosis of the infundibular tract, and furthermore the stenotic valve leaflets remain somewhat fused at their bases. In brief, we agree with Brock that the proper treatment of pulmonary valvular stenosis in association with the tetralogy of Fallot should consist of division of the valve, with the possible exception of those instances in which the condition is encountered in adults.

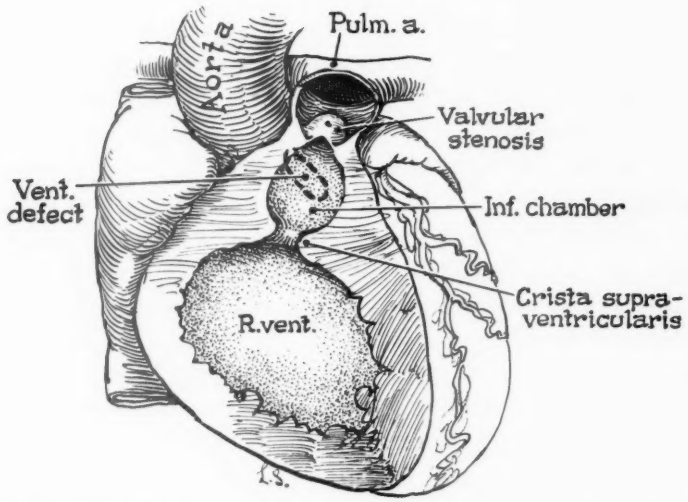

Fig. 6.—This drawing illustrates the combination of pulmonary valvular and infundibular stenosis in tetralogy of Fallot. Obviously both areas of stenosis must be dealt with in direct surgical procedures.

Before considering infundibular stenosis unaccompanied by valvular stenosis, we shall consider the combination of the two which was found in 15 per cent of our autopsy specimens. If the major point of obstruction is in the valve, it should be treated by division of the valve. If both obstructions are severe, it seems, in view of the fact that we have much to learn about the best treatment of at least one of the conditions and probably both, that the treatment should consist of an anastomosis between pulmonary and systemic arteries.

The results reported here show that there is a wide variation in the anatomy of infundibular stenosis. In some cases the obstructed area is short and in others it is long. In some cases it is high in the right ventricle and in others it is low. Some idea may be obtained preoperatively about the size of the infundibular chamber by radiologic studies. Direct observation of the area at the time of operation plus the use of pressure recording devices will give one a good idea of the nature, site, and degree of the stenosis. Even so, direct observation of the obstructed area in a bloodless field is impossible at present, and one has to use a good deal of imagination in visualizing what is being done in a direct attack on the stenotic area.

We shall now comment on the advantages and disadvantages of the direct attack in the treatment of infundibular stenosis as compared with an anastomotic procedure between pulmonary and systemic arteries. Brock[4] and Bailey, Glover,

and O'Neill[8] in stressing the advantages of the direct attack have stated that it does not add the danger of another anomaly, such as is caused by the creation of an artificial ductus. There may be considerable truth in this point of view; but may it not be that the traumatized area of myocardium denuded of its endocardium which results from infundibular resection may in itself present as great a hazard as does an additional anomaly in the form of an artificial ductus? In this connection it must be said that the patient with an artificial ductus rarely has subacute bacterial endarteritis or heart failure provided the ductus is of moderate size. Brock's statement that a successful direct attack on infundibular stenosis results in a lessening of the shunt of blood from the right side of the heart to the aorta appears to us to be a more important consideration. When the oxygen content of aortic blood is considerably higher than that in the pulmonary artery, the blood which passes directly to the lungs from the right side of the heart will gain more oxygen per unit volume than that transmitted to the lungs from the aorta. In other words, a given amount of work by the heart will result in a more effective oxygenation of the blood if it passes directly from the right heart to the lungs than if part of it is shunted through an interventricular defect, is admixed with blood of a higher oxygen content in the aorta, and then part of the mixture is shunted to the lungs. On the other hand, there is little difference in the oxygen content of blood in the aorta and in the pulmonary artery in many cases of the tetralogy of Fallot. This is particularly likely to be the situation when the aorta arises mainly from the right ventricle.

The disadvantages of the direct attack in the treatment of infundibular stenosis appear to be numerous at the present time although it is likely that some of these will be overcome with additional experience and technical and diagnostic advances. In the first place, the immediate operative mortality rate is higher in association with the direct attack, at least in our experience. There may be greater sudden blood loss during the operation. The heart usually withstands operations on the large blood vessels at its base better than it tolerates incisions into and manipulations within its cavities. Because the field is obscured by blood, infundibular resection is in the main a blind procedure. The results reported in this paper show that the morphology varies greatly and it is impossible in many cases to determine the exact nature of the malformation; hence one may remove part of the stenotic tract only to leave a constricted area behind. If an error is made, it is more likely to be in the direction of removing too little rather than too much tissue. Attention has already been called to the dangers of thrombosis and embolism in association with resection of part of the interior of the heart in contact with the circulating blood. An attack upon a pulmonary valve which is devoid of muscle presents less hazard in this regard. Furthermore, the direct attack is accompanied by some danger of injury to the aortic valves and their attachments.

A consideration which has caused us concern is connected with the decision as to the advisability of opening the pericardium in all or most cases in which an operation is performed for suspected pumonary stenosis. It is certainly true,

as Brock has emphasized, that the surgeon will increase his familiarity with the pathologic anatomy of this region if pericardial exploration is performed routinely. At the same time it must be admitted that this maneuver, with the associated examination of the area, lengthens the operating time and increases the postoperative complications.

We suspect that differences of opinion about the choice of operative procedures may be influenced somewhat by the ages of the patients with whom one is dealing. For example, we have the impression that our patients and those of Potts are of a younger average age than those of Brock and of Bailey. Certainly the heart of a child or an adult is more suitable for infundibular resection than that of an infant.

In concluding this discussion, we must agree with Brock that infundibular resection is theoretically preferable to the creation of an artificial ductus in the treatment of infundibular stenosis, but there are many patients in whom resection is impossible with the use of existing techniques and still others in whom it is inadvisable. If we may hazard a guess, we predict that for the next several years pulmonary atresia and high infundibular stenosis will be treated by the making of an artificial ductus, and that there will be an increase in the use of infundibular resection in the treatment of low infundibular stenosis with a large chamber. It is likely that the general adoption of infundibular resection even for this latter condition will not be rapid since the results with anastomosing procedures are in general very good and since most cardiologists interested in this field do not seem to view the direct attack with favor. The anatomic findings of Donzelot, d'Allaines, and associates[6] in a recent study of 54 cases of tetralogy of Fallot and the conclusions drawn therefrom are similar to those reported in this paper.

SUMMARY

An anatomic study has been made of the autopsy specimens of the heart and great blood vessels of 95 patients who had pulmonic stenosis or atresia accompanied by an interventricular defect. Cases of "pure" valvular stenosis with an intact ventricular septum were not included. An operation had been performed on 79 of the 95 patients in an attempt to relieve the disability and cyanosis. It is not claimed that these cases are representative of the condition of all of the approximately 1,100 patients upon whom such an operation was attempted, for it is apparent that the mortality rate is highest among those with the serious malformations.

The specimens were classified according to the anatomy of the pulmonary conus region. It became apparent that there is extreme variability in the bulbus cordis in the tetralogy of Fallot. The specimens may be divided into the following groups: (1) 23 per cent showed pulmonary atresia, (2) 10 per cent valvular pulmonary stenosis, (3) 15 per cent both infundibular stenosis and valvular stenosis, and (4) 52 per cent showed infundibular stenosis. This last group was subdivided as follows: (1) 37 per cent showed a low, well-developed infundibular chamber, (2) 37 per cent a high, small infundibular chamber, and (3) the chamber was intermediate in size and position in the remaining 26 per cent.

The type of operative procedure that should be employed in the treatment of these defects has been discussed in regard to choice between a direct attack and a shunting procedure. It is obvious that with the possible exception of adults a direct attack with division of the valve should be used in the treatment of stenosis of the valve and that an artificial ductus should be made in the treatment of pulmonary atresia. There is probably a justifiable difference of opinion regarding the choice of procedure in the treatment of infundibular stenosis. When resection techniques are further developed, it appears likely that low infundibular stenosis with a large chamber will be treated by a direct approach with infundibular resection, and that shunting procedures will continue to be used in most cases of high infundibular stenosis with a small chamber. The advantages and disadvantages of a direct attack and of a shunting procedure are discussed.

We are indebted to Drs. Bahnson, Sabiston, and Morrow, who aided in examining these specimens.

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UNSUITABILITY OF TRANSVENTRICULAR AUTOGENOUS SLINGS FOR DIMINISHING VALVULAR INSUFFICIENCY

THOMAS C. MOORE, M.D., AND HARRIS B. SHUMACKER, JR., M.D.,
INDIANAPOLIS, IND.

(From the Department of Surgery, the Indiana University School of Medicine)

IN VIEW of contemporary advances in both open and closed techniques of cardiac surgery, it has seemed desirable to evaluate the suitability of certain autogenous structures which might conceivably be used in cardioplastic procedures. Needless to say, it is of great importance that the fate of any material to be used as an intracardiac graft be studied carefully in animals prior to its use in human subjects.

In 1930, Wilson[7] reported experiments concerning the use of autogenous transventricular slings as a means of producing mitral stenosis. He used principally pericardial strips which he discovered ultimately became tough and inelastic. The sequelae of clotting and thrombosis were found to be the major hazards associated with the use of fascia lata. Clotting was not encountered, however, in the one dog in which a tendon was utilized.

Experience with the treatment of experimental mitral regurgitation by means of an inverted vein graft through the left ventricle was reported by Murray, Wilkinson, and McKenzie[3] in 1938. The sling graft was inserted through a cannula and was expected to act as a flutter tamponade which would plug the leak in the mitral "dike" during systole. Although only two of eight dogs survived, the authors felt that regurgitation was reduced. No information was given concerning the fate of these vein grafts. In 1950, Murray[2] stated that one graft was in good condition after seven years in a dog's heart. In ten patients he introduced a sling composed of inverted superficial vein through which a palmaris longus tendon had been inserted. Two of these patients died and eight were said to be in a fairly satisfactory condition.

The use of pericardium in the experimental replacement of resected valve leaflets under direct vision was described in 1949 by Templeton and Gibbon.[4] Although their early results appeared encouraging, the pericardial grafts were found to have become shrunken and fibrotic with the passage of time. They did not find thrombosis to be a problem. Bailey and his associates[1] reported in 1951 the use, in seven patients with mitral regurgitation, of a pedicle graft of pericardium which was described as having its blood supply maintained intact. The degree of regurgitation, as estimated by a finger in the auricle, was said to be reduced by at least 75 per cent at the time of operation, in six patients. No information was given concerning the fate of these grafts or the mortality rate associated with their use. They had found free grafts to be ineffectual in animals and felt that better results were obtained with pedicle grafts of pericardium. However, no details of this experimental work were given. It would appear that certain criteria of adequacy and safety should be fulfilled by autog-

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enous slings before their use in the ventricles of human beings can be justified. The tissue should not precipitate thrombus formation nor should it interfere with cardiac action. It is desirable that it should maintain its structure, viability, and resilience. In addition, it should do the job for which it is intended and be able to withstand the constant trauma of cardiac activity without disabling fibrosis, degeneration, or rupture. It was to investigate the suitability of various transventricular autogenous tissue slings that this study was undertaken.

METHODS

Healthy mongrel dogs were used. The five types of autogenous tissue selected for use as slings were tendon, artery, vein, posterior rectus fascia covered by peritoneum, and pericardium. Their selection was based on strength, pliability, and the presence of endothelial or mesothelial surface which might lessen the likelihood of thrombus formation. The long tendon employed was that of the extensor carpi radialis. Posterior rectus abdominis fascial strips were made into slings by rolling them into tube form with the outer surface covered by the mesothelium of the parietal peritoneum. Pericardial strips were similarly converted into mesothelial-covered tubes, both free and pedunculated grafts being used. The long artery and vein grafts were obtained from the iliofemoral area. The vessels were inverted prior to use so that the outside surface would be completely endothelialized. Strict surgical asepsis was observed at all times.

Pentobarbital sodium induction with controlled oxygen and ether anesthesia through an endotracheal tube was used. The left chest was entered through an intercostal incision and the pericardium was opened anterior to the phrenic nerve. Prior to insertion of the sling, the heart was steadied by tension on a traction suture through its apex. One end of the sling was secured to the blunt end of a large "autopsy" needle and a traction suture was placed through the other end. A silk pull-out suture was placed in the mid-portion of the sling so that the slack of the graft could be left within the ventricle in order to permit the sling to have potential flutter tamponade action. The needle was thrust through the ventricle and both ends of the sling were sutured to the ventricular walls with silk sutures. The pericardium was left open when a sizable pericardial defect was created. Otherwise, it was closed loosely. The chest incision was repaired with interrupted silk sutures and the pleural cavity was aspirated at the completion of closure. Blood loss was minimal and there were no deaths during operation or the early postoperative period. Penicillin was given routinely following operation.

RESULTS

Inverted arterial slings were used in two dogs (Table I). Although the use of autogenous arterial slings would not be feasible in human beings, it was felt that the fate of the autogenous arterial grafts in dogs would certainly indicate the optimum that could be expected from the intraventricular utilization of arterial homografts. The dog in which the inverted arterial sling had been

placed across the right ventricle was examined forty-nine days following operation. The sling was intact without evidence of thrombus formation. It had, however, become inelastic, fibrotic, and moderately contracted. When the other dog was sacrificed seventy-nine days postoperatively, the left ventricular arterial sling was found to have ruptured. The remnants had retracted to form two relatively small swellings on the endocardium. Microscopic study of one of these areas revealed considerable intimal proliferation of the remnant with central degeneration. It is of interest that the degeneration, which was continuing in the myocardium of this dog, was cellular, whereas that of the intact slings to be described later was of an acellular and presumably ischemic type. The infiltration was predominantly of plasma cells.

TABLE I. SUMMARY OF RESULTS WITH TRANSVENTRICULAR AUTOGENOUS SLINGS IN FOURTEEN DOGS

DOG	NATURE OF SLING	VENTRICLE	DURATION OF PLACEMENT (DAYS)	STATUS OF SLING	THROMBUS FORMATION	FIBROSIS	DEGENERATION
1.	Inverted artery	Right	49	Intact	0	++	+++
2.	Inverted artery	Left	79	Ruptured	0	+++	++++
3.	Inverted vein	Right	83	Intact	0	++	++++
4.	Inverted vein	Right	88	Intact	0	++	++++
5.	Inverted vein	Left	29	Intact	0	+++	++++
6.	Inverted vein	Left	91	Intact	0	+++	++++
7.	Tendon	Right	105	Intact	0	++	++++
8.	Tendon	Right	106	Intact	0	+++	++++
9.	Tendon	Left	104	Intact	0	+++	++++
10.	Tendon	Left	95	Intact	0	+++	++++
11.	Pericardium (free)	Right	11	Intact	+++	+	++
12.	Pericardium (free)	Left	98	Intact	+	+++	++++
13.	Pericardium (pedicle)	Left	26	Intact	0	+++	+++
14.	Pericardium (pedicle)	Left	70	Intact	0	++++	+++
15.	Pericardium (pedicle)	Left	58	Ruptured	0	++++	+++
16.	Pericardium (pedicle)	Left	58	Intact	0	++++	++++
17.	Pericardium (pedicle)	Left	57	Intact	0	++++	+++
18.	Pericardium (pedicle)	Left	44	Intact	0	+++	+++
19.	Rectus Fascia-Peritoneum	Right	20	Ruptured	++++	++	++
20.	Rectus Fascia-Peritoneum	Left	92	Intact	++	+++	++++

Transventricular inverted veins were used in four dogs, through the left ventricle in two, and the right in two. The results were surprisingly uniform, varying principally in the degree of alteration. The extent of structural change was influenced both by the duration of placement and the ventricle traversed. The trauma to which a sling in the left ventricle was subjected was great and, as one might have expected, the degree of damage to the sling encountered, for

approximately equal periods of placement, was more pronounced on the left than on the right side of the heart. The vein slings were all fibrotic and contracted with loss of elasticity; the changes were more marked when the vein was placed in the left ventricle (Fig. 1). None of them showed either gross or microscopic evidence of thrombus formation. The more marked contracture of the left ventricular slings appeared to produce a drawing together of the

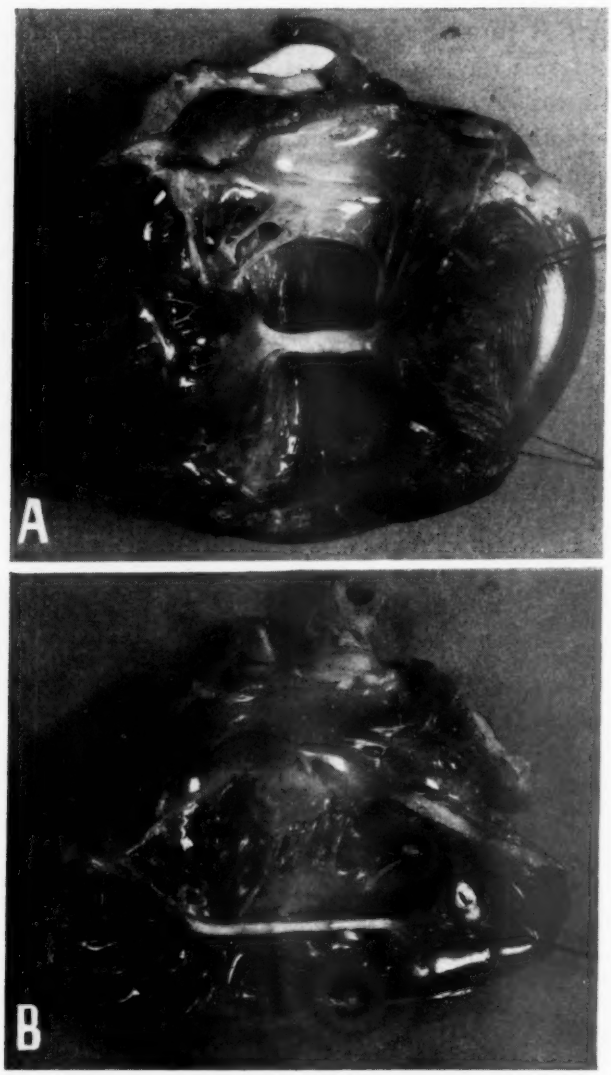

Fig. 1.—Photographs of intraventricular inverted vein slings (*A*) across the left ventricle of Dog 6 and (*B*) across the right ventricle of Dog 4.

ventricular walls with a limitation in the capacity and distensibility of the ventricle. Microscopic study of the inverted vein segments following their removal from the ventricle showed marked intimal proliferation in all. Hematoxylin and eosin stains also demonstrated the media and adventitia to be structureless and acellular. These areas seemed to have undergone a profound

ischemic degeneration. Fragmentation and vacuolization of the amorphous central areas were encountered in some sections. Sections of the slings near their junction with the myocardium tended to show some capillary invasion of the degenerating graft. Verhoeff's[6] elastic tissue stain, with Van Gieson's[5] counterstain, demonstrated the amount of residual elastic tissue to be greater in the right ventricular sling (Fig. 2). In addition, the remaining elastic tissue appeared to be thrown up in folds by the increasing circular constriction secondary to intimal proliferation and fibrosis. Intimal and medial zones were clearly demarcated and intimal proliferation was great. Progressive fragmentation and gradual disappearance of the elastic tissue were evident.

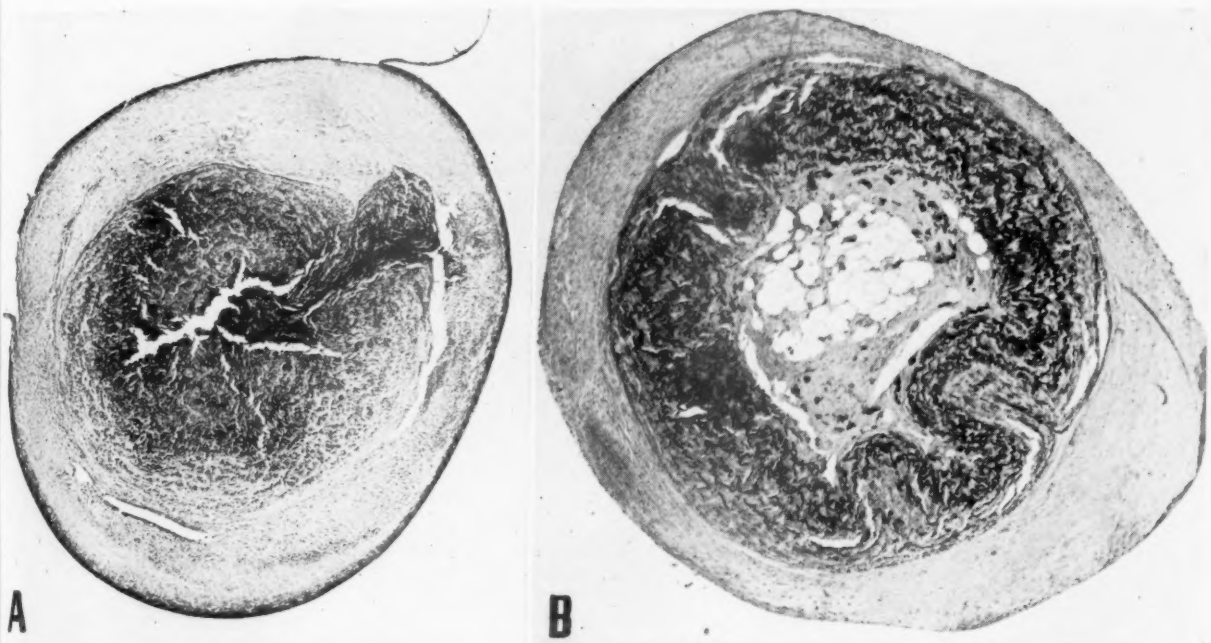

Fig. 2.—Photomicrographs of vein slings pictured in Fig. 1 showing more marked intimal proliferation and elastic tissue degeneration (*A*) in Dog 6 than (*B*) in Dog 4 (Verhoeff-Van Gieson stain).

Tendon transplants were carried out on four dogs. The tendons remained intact, and there was no thrombus formation. Such thickening and fibrosis were encountered as to transform these tendons into rigid and inelastic structures. Some hard surface nodules were present and there was also a tendency toward superficial vascular invasion from the myocardium (Fig. 3). The central portion of all four tendons showed the complete loss of both cells and structure. All that remained was a relatively homogeneous hyaline stroma. Some peripheral zones showed fibroblastic proliferation of connective tissue. Even these areas were relatively acellular. Sections through the nodular areas showed significant cartilaginous metaplasia, presumably on the basis of the trauma encountered (Fig. 4). The occasional cells still present in such areas were separated into lacunae surrounded by a basophilic homogeneous substance.

Fig. 3.—Photograph of tendon sling through right ventricle of Dog 8 demonstrating nodularity and thickening.

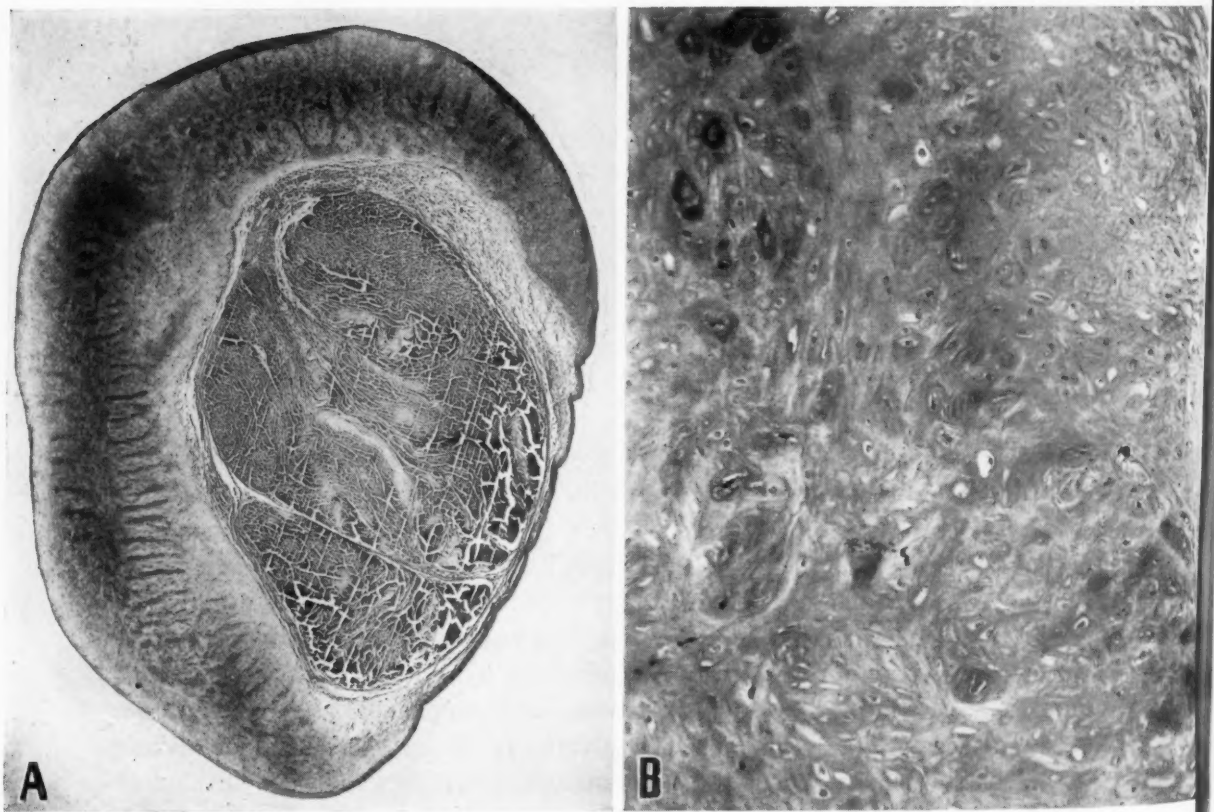

Fig. 4.—High- and low-power photomicrographs illustrating central degeneration and fragmentation and peripheral cartilaginous metaplasia of tendon from Dog 8 (hematoxylin and eosin).

The results with free grafts of pericardium were even less satisfactory. One dog died eleven days following operation. When the right ventricle was opened, the sling was found to have become markedly enlarged with the formation of a large firm thrombus near one end (Fig. 5). The sling had increased both in size and rigidity. Microscopically, there was a large thrombus with fibrosis and beginning organization. The rolled pericardium was undergoing degeneration but its form was still intact. It was evident that some fat had not been removed prior to the placement of the sling because there were occasional areas of fat necrosis and vacuolization (Fig. 6). The dog with a pericardial sling through the left ventricle was examined ninety-eight days postoperatively. Although the sling was intact, it was markedly fibrotic, inelastic, and contracted.

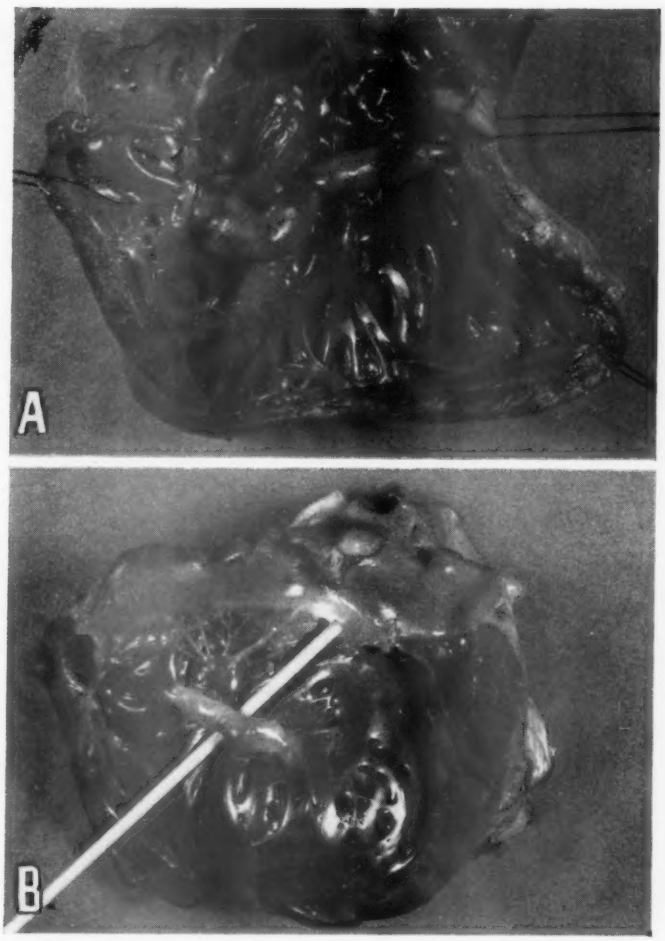

Fig. 5.—Photographs of free pericardial slings in (*A*) Dog 11 and (*B*) Dog 12.

Microscopic study revealed the presence of a slowly organizing thrombus at one circumferential periphery and fibroblastic proliferation at the other. The central area was completely acellular and amorphous.

Pedicle slings of pericardium, prepared in the manner described by Bailey and his associates, were employed in six dogs. The findings were essentially

the same as those encountered with the use of free pericardial grafts. Although thrombus formation was not encountered, all six slings became grossly thickened, fibrotic, and contracted, and all showed microscopic evidence of ischemic degeneration. One sling ruptured and one was found to have histologic changes similar to the cartilaginous metaplasia encountered in one of the previously described tendon slings.

One dog with a posterior rectus fascial sling through the right ventricle died twenty days following operation. Death was found to be due to interference with tricuspid valvular action due to a large thrombus which had formed about one end of the ruptured and retracted sling. The other rectus fascial sling, which had been placed through the left ventricle, was examined ninety-five days following operation. It was markedly fibrotic and contracted and was found on microscopic examination to have undergone ischemic acellular degeneration. There was evidence of a slowly organizing thrombus. Thus, of the twenty dogs, two died of the sequelae of thrombus formation and two presumably of pneumonia. The remaining dogs were sacrificed.

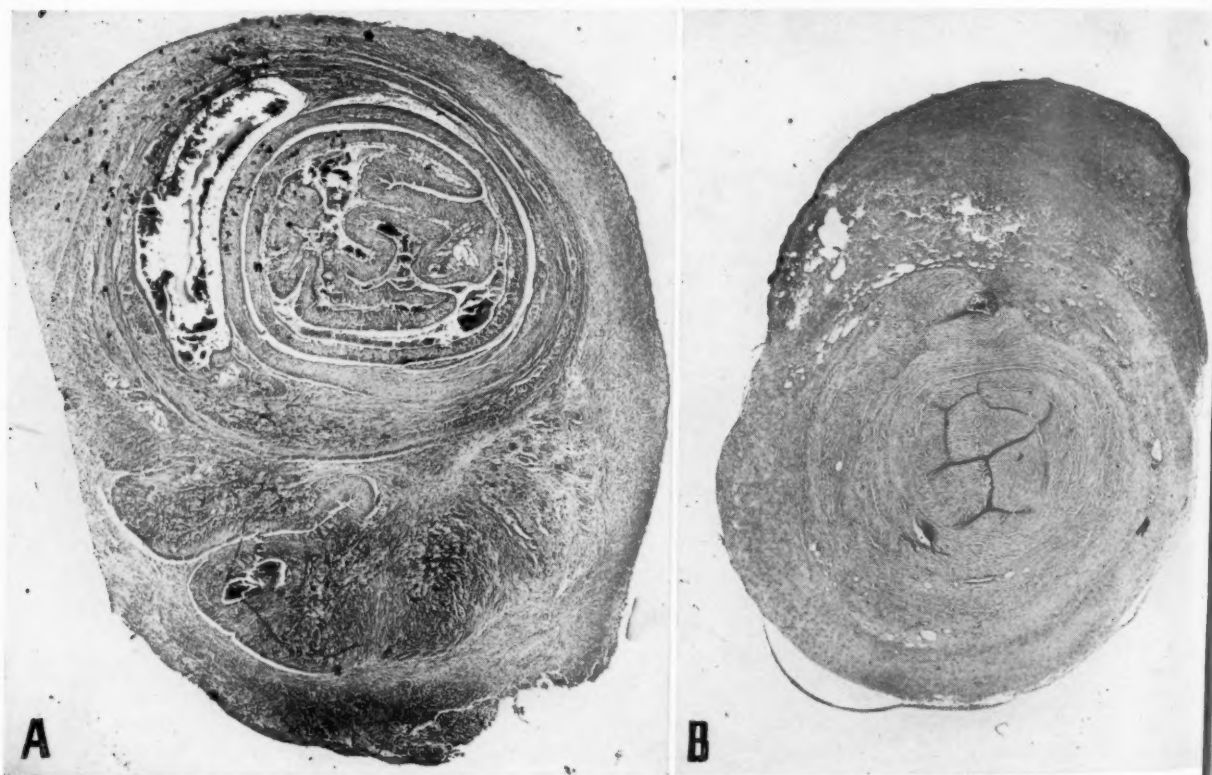

Fig. 6.—Photomicrographs of pericardial slings showing thrombus formation and pericardial degeneration with some fat necrosis in (*A*) Dog 11 and (*B*) Dog 12.

DISCUSSION

If tamponade of a defective mitral or tricuspid valve by a transventricular sling were to be an effective means of reducing valvular insufficiency, the success of this procedure would depend upon the selection of a type of sling which could

withstand the considerable trauma to which it would be constantly subjected without losing its shape and elasticity and without rupturing or precipitating thrombus formation. All of the autogenous tissues used became grossly fibrotic and contracted within relatively short periods of tme. It was quite apparent that they had all become functionally ineffectual. It addition, thrombus formation seemed to accompany the use of free grafts of pericardium and posterior rectus abdominis fascia covered by peritoneum. The degree of rigidity and contracture, for equal periods of time, was noted to be greater when the sling was across the left ventricle than when it was through the right. This finding suggests that the greater trauma associated with the higher left ventricular pressures was a significant factor in the determination of the extent of sling alteration.

On microscopic examination the gross changes in the slings were found to be due to an extensive central degeneration of an acellular and ischemic nature associated with a small peripheral zone of fibrosis. The slings characteristically lost their distinguishing structural features and their central portions assumed an amorphous, homogenous appearance. The absence of vascular and cellular infiltration was conspicuous. Even the organization of the thrombi which occasionally formed was slow and sluggish as evidenced by the absence of the usual capillary and cellular infiltration. It was evident that these free grafts did not survive and that the failure of survival was due to the lack of effective vascularization and to the constant trauma and motion to which they were subjected in an environment to which they were ill suited.

The result was a fibrotic, rigid, and amorphous ghost of the transplanted tissue which obviously could not be expected to provide effective flutter tamponage for the reduction of valvular insufficiency.

Furthermore, the use of a so-called pedicle graft of pericardium which might, theoretically, carry an adequate blood supply of its own has been disappointing. Our experience with pedunculated pericardial grafts indicates that it is unlikely that a transventricular sling of this type will remain viable and functional on the basis of its own blood supply. Although the degree of acellularity and ischemic degeneration was less marked in pedicle than in free slings, these microscopic changes were present, and the gross alterations of the pedicle slings were indistinguishable from those encountered with the use of the various types of free grafts. Anatomic limitations of this procedure have been encountered. The arterial blood supply of the pericardium is maintained by a series of small vessels which generally run in an essentially horizontal direction. The posterior portion of the pericardium is supplied by pericardial branches from the thoracic aorta while the anterior portion receives its arterial blood from pericardial branches of the internal mammary and musculophrenic arteries. Most of the anterior vessels are located in the thick fat pad which is adjacent to the pericardium of this area. This sizable fat pad cannot be sacrificed without interruption of the blood supply, and its inclusion with the sling predisposes to massive fat necrosis from intraventricular trauma. Furthermore, the pedicle incision advocated by Bailey and his associates was parallel and anterior to the phrenic nerve. Such an incision would therefore be across,

rather than parallel to, the principal vessels supplying the pedicle. They also stated that blood loss from the myocardial incisions would be prevented by the tamponade effect of the sling in these areas. In this regard, it would appear that tamponade of the tenuous and highly questionable blood supply of this long pedicle might also occur. Accordingly, we feel that the successful use of a pericardial pedicle sling would be seriously compromised by the inadequacy of its blood supply, by myocardial constriction of its neck, by the substantial trauma of ventricular activity, and by the necessary length of the sling to be effective. Our experience with the experimental use of these slings would appear to confirm these impressions.

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HOMOLOGOUS ARTERIAL GRAFTS AND AUTOGENOUS VEIN GRAFTS USED TO BRIDGE LARGE ARTERIAL DEFECTS IN MAN

A Report on Fourteen Cases

Francis N. Cooke, M.D.,* Miami, Fla., and
Lieutenant Colonel Carl W. Hughes, Medical Corps, United States Army,
Captain Edward J. Jahnke, Medical Corps, United States Air Force, and
Brigadier General Sam F. Seeley, Medical Corps, United States Army,
Washington, D. C.

(From Walter Reed Army Hospital, Washington, D. C.)

FOLLOWING the outbreak of hostilities in Korea, a large number of the wounded with vascular injuries have been admitted to the Army Vascular Center at Walter Reed Army Hospital for treatment. A report on the management of 101 such cases was made in October, 1951. At that time we reported that arterial continuity had been successfully re-established in 54.7 per cent of the major vessels with only one technical failure. Since Jan. 1, 1951, it has been our policy to re-establish the continuity of all major arteries, either by suture anastomosis of the damaged artery, or, if the defect by virtue of its size precluded approximation of the vessel, a graft has been used to bridge the defect. Of fifty-six major vessels operated upon during this period, vascular continuity has been re-established in fifty-five or 98.2 per cent. The use of grafts in the surgical management of vascular injuries has been an important adjunct to our armamentarium. It has long been established[2-13] that homologous arterial grafts and autogenous vein grafts can be successfully used and will be initially effective in restoring vascular continuity. The fate of such grafts, however, has been a controversial subject. The purpose of this presentation is to report the initial results and early follow-up on the cases in which vascular grafts were used in Korean war wounded.

As previously reported,[1] it has been our policy, whenever possible, to delay definitive surgery three months from the time of wounding to allow collateral circulation to adjust to the increased demands. In most cases, collateral circulation was shown by the reactive hyperemia test to be sufficient to maintain viability of the extremity should the vessels at the site of injury be completely occluded. This allowed us a margin of safety should thrombosis and occlusion occur at the operative site.

METHODS

With adequate exposure of the lesion, the artery above and below the false sac or fistula is occluded with Potts clamps. We have found the Potts ductus clamps especially useful in this work. After the lesion has been excised, the artery is carefully inspected to assure that all scar tissue has been cut away

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*Formerly Assistant Chief, Surgical Service, Walter Reed Army Hospital.

and normal arterial wall remains. If the defect in the artery is small, it is closed by suture and if the vessel has been transected, an end-to-end anastomosis is performed. Should the defect be too large for approximation of the vessel, a graft is used.

The vessels are sutured in the following manner: Two everting mattress sutures, placed on opposite sides of the vessels, are used as stay sutures. A continuous everting mattress suture completes the anastomosis (Fig. 1). In all cases we have carefully sought an intima-to-intima approximation. Anticoagulants have not been used. Suture repair of veins in the extremities has not been practiced.

Five of the grafts used were homologous arterial grafts from the Walter Reed artery bank,[14] and nine were autogenous vein grafts taken at the time of surgery. Of the five cases in which preserved homologous arterial grafts were used, two have shown occlusive changes.

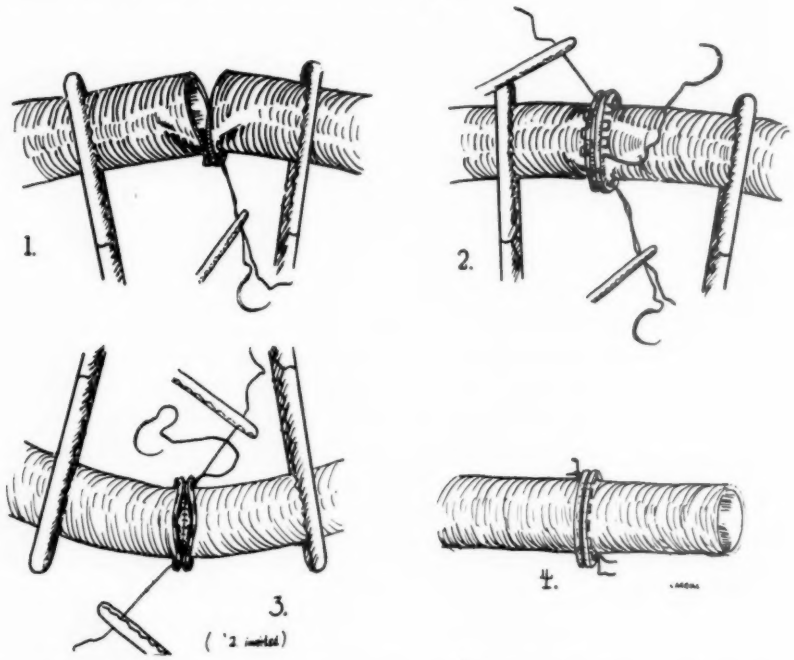

Fig. 1.—Two everting mattress sutures placed on opposite sides of the vessels are used as stay sutures. The anastomosis is completed with a continuous everting mattress suture.

CASE 1-A.—The patient had an aneurysm of the left axillary artery, resulting from a gunshot wound in Korea in September, 1950. After excision of the aneurysm a 4 cm. homologous arterial graft which had been preserved in modified Tyrode's solution was used to bridge the defect. Blood flow through the graft was immediately apparent. Four months later the patient was sent to duty with the radial pulses equal. Follow-up reports indicated that the pulsations in the left wrist had decreased. The patient was readmitted to Walter Reed Hospital at our request. Pulsations in the affected arm were less to palpation, and oscillometric readings were decreased. It was concluded that the graft had undergone occlusive changes approximately seven months after reconstructive surgery. Despite this fact, the patient was returned to duty with no apparent impairment of function and is now overseas. The latest follow-up was at ten months and the graft has been in place eighteen months.

TABLE I

ARTERY	HOMOLOGOUS ARTERY GRAFTS	AUTOGENOUS VEIN GRAFTS
Subclavian	1	0
Axillary	1	2
Brachial	1	1
Common Femoral	1	1
Superficial Femoral	0	3
Popliteal	1	2
Total	5	9

Case 2-A.—The patient had an arteriovenous fistula of the left subclavian vessels, resulting from a gunshot wound in Korea. In addition, a complete brachial plexus palsy resulted from the injury. After excision of the fistula, the defect in the artery was bridged by a homologous artery graft twelve days old. Following surgery the pulse in the affected wrist returned to full volume and became equal to that of the uninjured extremity. During the ensuing weeks the patient had recurring attacks of malaria. Four months later it was noted that his arm was cool, and oscillometry was decreased. The radial pulse had disappeared. It was assumed that occlusion had occurred approximately four months after operation.

Case 3-A.—Traumatic anueurysm of the right brachial artery resulted from shell fragment wounds in Korea. The aneurysm was excised and the defect bridged by a 7 cm. 3-day-old homologous artery graft. Following surgery, pulsations in the wrist and over the graft have remained equal to that on the opposite side. Attempted angiograms were not successful. However, radial pulsations and blood pressure readings have not changed over a seven-month follow-up period.

Case 4-A.—The patient had a traumatic arteriovenous fistula of the left popliteal vessels, a result of enemy action in Korea. The fistula was excised and the defect bridged with a homologous arterial graft 5 cm. long and eighteen days old. An arteriogram five months later showed good function of the graft. The patient was returned to duty. No change in pulses or clinical signs have occurred over a twelve-month follow-up period.

Case 5-A.—Traumatic arteriovenous fistula of the left common femoral vessels resulted from mortar wounds. The arteriovenous fistula was excised and a homologous arterial graft was used to bridge the defect. An aortogram five months later demonstrated a patent graft with good function. The patient was returned to duty and has since been separated because of expiration of enlistment. Over an eleven-month follow-up period, clinical signs have not changed.

Nine of the cases which comprise this report have had autogenous veins used to bridge arterial defects.

Case 1-V.—Traumatic aneurysm of the left popliteal artery resulted from gunshot wounds in Korea. The aneurysm was excised and a section of saphenous vein inserted as a graft. Arteriograms four months later showed excellent function of the graft. A follow-up period of nine months has shown no changes.

Case 2-V.—The patient suffered traumatic aneurysm of the left superficial femoral artery when wounded in Korea by hand grenade. The aneurysm was excised and a 4 cm. section of saphenous vein was grafted into the arterial defect. Pulsations in the foot have been excellent. The patient was returned to duty. During a ten-month follow-up period, clinical signs have not changed.

CASE 3-V.—The patient suffered a traumatic aneurysm of the left popliteal artery due to enemy action in Korea. The aneurysm was excised and the defect bridged with a segment of the popliteal vein. Arteriograms three months postoperatively showed a slightly dilated

Fig. 2 (Case 4-A).—Homologous artery graft used to bridge a defect in the popliteal artery.

Fig. 3. Fig. 4.

Fig. 3 (Case 4-A).—Arteriogram demonstrating patent graft five months after insertion.

Fig. 4 (Case 3-V).—Arteriogram taken three months postoperatively demonstrates patent autogenous vein graft bridging a defect in the superficial femoral artery.

but functioning graft. The patient was returned to duty. Clinical signs did not change during the three months of observation.

Case 4-V.—Traumatic arteriovenous fistula of the superficial femoral vessels resulted from wounds received in Korea. The fistula was excised and a segment of the saphenous vein utilized to bridge the defect. Arteriograms revealed a functioning graft. Clinical signs have remained unchanged during the three months before return to duty.

Case 5-V.—Traumatic arteriovenous fistula of the left brachial artery and vein resulted from a gunshot wound. The fistula was excised and the defect bridged by a graft from the basilic vein. Excellent pulsations continued following surgery. The follow-up period was only three months.

Fig. 5 (Case 4-V).—Arteriogram of an arteriovenous fistula of the superficial femoral vessels.

Case 6-V.—Traumatic arteriovenous fistula of the superficial femoral vessels resulted from wounds received in Korea. The fistula was excised and a segment of the saphenous vein was used to bridge the defect. A large false sac which had contained 1,500 c.c. of blood and clot became infected postoperatively, necessitating extensive drainage incisions. The graft functioned well despite the complications. A postoperative arteriogram showed the reconstructed arterial channel to be constricted. Nine months later a repeat arteriogram demonstrated complete occlusion at the graft site.

Case 7-V.—Traumatic aneurysm of the right axillary artery resulted from a gunshot wound. The aneurysm was excised and the defect bridged with a 5 cm. section of saphenous vein. Following operation the graft appeared quite small and pulsation below the operative site was feeble. However, in the postoperative period pulsation in the brachial artery was evident and a pulse in the radial artery was present, but weak. No arteriograms were obtained. The patient was returned to military duty after two months with a fair pulse. Poor judgment was used in this case in using a graft too small for the artery, and in failure to remove the graft and insert a larger one when this was realized at the time of surgery.

Case 8-V.—Traumatic aneurysm of the right common femoral artery occurred when the patient was wounded in action in Korea. This case has been previously reported,[1] and is mentioned here to complete this series. On admission, dorsalis pedis and posterior tibial pulsations were good. The aneurysm suddenly began to enlarge. Distal pulses disappeared and the foot assumed a deathlike pallor. Emergency operation was performed and the aneurysm excised. There was no back flow from the superficial femoral artery suggesting

thrombosis or spasm of that vessel distal to the operative field. A fine catheter was passed down this vessel and gentle suction was applied. This failed to relieve the situation. The profunda artery had a brisk back flow. A saphenous vein graft bridged the defect in the common femoral artery, re-establishing blood flow from the common femoral to the profunda femoris artery. The leg became warm, but pulsations in the dorsalis pedis and posterior tibial arteries were absent, which was to be expected because of the demonstrated occlusion of the superficial femoral artery. Unfortunately, angiograms were unsuccessful, although two attempts were made. Later, dorsalis pedis and posterior tibial pulses returned in full volume. The patient was separated from the service eleven months postoperatively because of associated disabling injuries.

Fig. 6 (Case 4-V).—Arteriogram showing a patent autogenous vein graft in the superficial femoral artery.

Case 9-V.—Traumatic aneurysm of the left axillary artery occurred. After excision of the lesion a section of the cephalic vein was used to bridge the defect. Distal pulses have been excellent. There has been a follow-up of only one month.

SUMMARY AND CONCLUSION

Fourteen cases of traumatic aneurysms and arteriovenous fistulas of major vessels have been presented in which vascular continuity could not have been re-established by suture repair or direct anastomosis. Homologous arterial grafts were used to bridge the arterial defect in five cases. Occlusive changes occurred at the graft site in two of these cases, one after four months and the

other after seven months. Autogenous vein grafts were used in nine of the fourteen cases. At the present time eight autogenous grafts appear to be functioning well; one graft thrombosed following a severe postoperative infection.

It is admitted that the evidence as to the adequacy of function in all cases is not incontrovertible. The period of follow-up is relatively short, the longest being only twelve months. However, we are of the opinion at the present time that, excluding the aorta, the use of autogenous vein grafts in our hands offers the best method of restoring the continuity of major arteries in which large defects exist.

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THE INFLUENCE OF DIAMETER DISPROPORTION AND OF LENGTH ON THE INCIDENCE OF COMPLICATIONS IN AUTOGENOUS VENOUS GRAFTS IN THE ABDOMINAL AORTA

EVERETT J. SCHMITZ, M.D.,* EDMUND A. KANAR, M.D.,*
LESTER R. SAUVAGE, M.D.,** EDWARD H. STORER, M.D.,*
AND HENRY N. HARKINS, M.D.,*** SEATTLE, WASH.

(From the Department of Surgery, University of Washington School of Medicine)

THE surgeon faced with the necessity of using a graft to bridge an arterial defect is interested in the functional properties of available types of grafts. From a practical standpoint, preserved arterial homografts and fresh venous autografts appear to be the most attractive. It is our purpose in this report to assess some of the properties of fresh venous autografts in both the adult and the growing animal.

There are three principal factors which lead to thrombosis in a recently implanted vascular graft: (1) injury of the intimal surface, (2) protrusion of foreign bodies (such as sutures) into the blood stream, (3) presence of excessive turbulence in blood flow. In order to have a clear understanding of the last named factor, we have used a classification of various grafts on the basis of graft-aortic disproportions. The site of maximum turbulent flow in certain of these grafts is diagrammed in Fig. 1.

With normal flow of blood, Smith (1945) has demonstrated a moving central core of cellular blood elements and a peripheral clear zone. So long as these two zones remain distinct there is little tendency toward clotting. However, when a turbulent flow is present this zonal flow is disrupted and the cellular elements come in contact with the inner surface of the vessel, increasing thereby the possibility of thrombosis. In a vascular graft the danger of thrombosis is greatly magnified in the presence of turbulent blood flow since there is usually some suture material exposed as well as minute points of intimal damage at the point of anastomosis and, at a later period, there may be larger areas without satisfactory endothelial covering.

In the experimental data to be presented, the grafts used were chiefly those which were isodimensional, with biterminal negative disproportion or with biterminal positive disproportion.

METHODS OF PROCEDURE AND EXPERIMENTAL RESULTS

The experimental data were obtained from work on mature dogs and growing pigs and the presentation is separated accordingly.

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*Research Associate in Surgery.

**Research Associate and Damon Runyon Fellow in Surgery.

***Professor of Surgery.

I. *Fresh Venous Autografts Implanted in the Abdominal Aorta of Mature Dogs.*—Forty healthy mongrel dogs having an average weight of 16.4 kilograms (range 9.8 to 30.0 kilograms) were anesthetized with intravenous Nembutal and the abdominal aorta was exposed by a midline transperitoneal approach. All operative procedures were performed under strict aseptic conditions. The length of the excised segment of aorta involved a ratio of approximately 1 cm. to each 1.5 cm. of venous graft to be implanted. The fresh autografts used were seventeen external jugular veins and the remainder were inferior caval segments, a few of the latter having their diameters altered by a method of plication to be described later. Anastomoses were performed for the most part using a continuous Carrel-type suture of 00000 black silk on atraumatic needles. A few

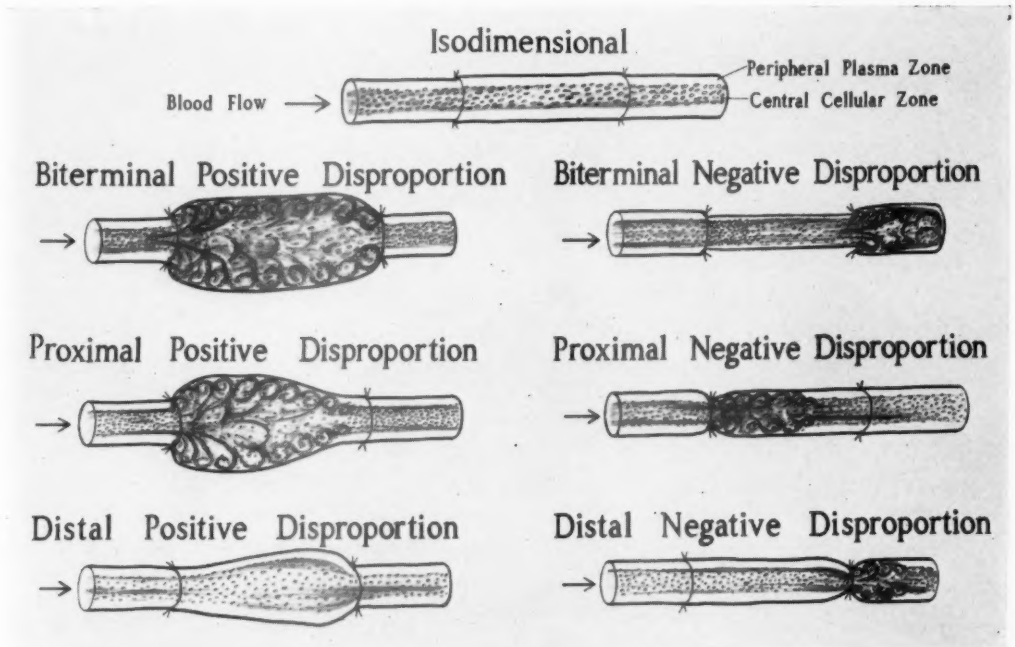


Fig. 1.—A classification of vascular grafts on the basis of graft-aortic disproportions with their associated hemodynamic disturbances. Isodimensional grafts have no disturbance of blood flow within them. In the grafts with disproportion, it appears that the grafts with distal positive disproportion have the least alteration in the stream of flow. Maximum turbulent flow occurs in grafts with biterminal positive disproportion.

anastomoses were performed using a continuous everting mattress type of suturing. After completion of the anastomoses, suitable calipers were used to obtain measurement of the graft length, graft diameter, and diameter of the aorta 1 cm. above and 1 cm. below the graft. Special attention was directed toward avoidance of any desiccation of these fresh grafts during the operative procedure. Each dog received 200,000 units of penicillin daily during the first three postoperative days and was kept in a heated paddock in the laboratories for two weeks following graft implantation. Vascular complications developing during this period are herein designated "early complications." Thereafter, dogs were kept at a boarding farm and returned to the laboratories for study whenever necessary. Four immediate postoperative deaths not related directly to the graft

are eliminated from further consideration, the remaining thirty-six animals being considered as providing valid data for the discussion below.

In Table I are given the results found regarding the early complications in fresh venous autografts of the abdominal aorta in mature dogs. There were fourteen deaths in the early postoperative period, but two of these dogs were sacrificed while in good health with their venous grafts open for purposes of study in another group of vascular experiments. Six of the vascular deaths were due either to rupture of the vein graft or dehiscence of the suture line, and six additional deaths were caused by early occlusive thrombosis of the graft. One of the thrombosed grafts was an inferior vena caval segment 54 per cent larger in diameter than the recipient aorta and 7.4 cm. long. This animal (R-100) was saved for later study by having the thrombosed graft excised and replaced by an external jugular vein graft 7 per cent larger in diameter than the aorta and 10.8 cm. in length. This latter graft was open at the sacrifice of the dog twenty-three weeks after implantation.

TABLE I. THE INCIDENCE OF EARLY COMPLICATIONS IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN MATURE DOGS

Total number of fresh venous grafts	36	100%
Deaths due to early graft complications	12*	33.3%
Early thrombosis of grafts	7†	19.4%
Anastomosis dehiscence	4	11.1%
Ruptured graft	2	5.6%

*Two nonvascular deaths not included.
†One of these animals did not die.

In order to evaluate the factors of graft length and biterminal positive disproportion (Fig. 1) in early thrombosis, we studied a series of eighteen mature dogs having short-length grafts (average length 2.64 cm., range 2.2 to 3.05 cm.), long-length grafts (average length 8.37 cm., range 6.5 to 10.8 cm.) and all with some degree of positive disproportion. The average diameter of the aorta in this group was 0.73 cm. (range 0.60 to 0.99 cm.) while the average diameter of the grafts was 1.12 cm. (range 0.75 to 1.60 cm.). Table II gives the incidence of early thrombosis on the basis of length of the autogenous vein grafts having positive disproportion.

TABLE II. THE INFLUENCE OF GRAFT LENGTH AND BITERMINAL POSITIVE GRAFT-AORTIC DISPROPORTION UPON EARLY THROMBOSIS

DESCRIPTION	TOTAL NO. OF DOGS	NO. OF THROMBOSES	PER CENT OF THROMBOSIS
Grafts longer than 5 cm.	12	7	58.3
Grafts shorter than 5 cm.	6	0	0

However, more significant than the simple consideration of graft length with variable degrees of positive graft-aortic disproportion is the examination of the data on the basis of length and *magnitude* of the positive graft-aortic disproportion as tabulated in Table III with reference to the frequency of early thrombosis. There was a high incidence of early thrombosis in fresh venous autografts which were considerably larger than the recipient aorta and longer than 5.0 cm. in length.

TABLE III. THE INFLUENCE OF GRAFT LENGTH AND MAGNITUDE OF POSITIVE GRAFT-AORTIC DISPROPORTION UPON EARLY THROMBOSIS

DESCRIPTION	TOTAL NO. OF DOGS	NO. OF THROMBOSES	PER CENT OF THROMBOSIS
Grafts *1 to 20 per cent* larger and over 5 cm. long	3	0	0
Grafts *21 to 100 per cent* larger and over 5 cm. long	9	7	77.7
Grafts *21 to 100 per cent* larger and under 5 cm. long	6	0	0

The vein grafts which were isodimensional or had biterminal negative disproportion at the time of anastomosis in the animal showed no early thrombosis. This series of eighteen additional animals included grafts of short length (average 3.52 cm., range 1.8 to 4.9 cm.) and long length (averages 8.01 cm., range 5.85 to 10.30 cm.). The isodimensional grafts had an average diameter of 0.69 cm. (range 0.55 to 0.80 cm.). The grafts with negative disproportion had an average diameter of 0.58 cm. (range 0.45 to 0.80 cm.) as compared to an average aortic diameter of 0.73 cm. (range 0.55 to 1.10 cm.). As shown in Table IV, early thromboses did not occur with these grafts.

TABLE IV. THE INFLUENCE OF GRAFT LENGTH AND BITERMINAL NEGATIVE GRAFT-AORTIC DISPROPORTION OR ISODIMENSIONAL GRAFT-AORTIC PROPORTIONS ON EARLY THROMBOSIS

DESCRIPTION	TOTAL NO. OF DOGS	NO. OF THROMBOSES	PER CENT OF THROMBOSIS
Grafts longer than 5 cm.	11	0	0
Grafts shorter than 5 cm.	7	0	0

In a series of four inferior vena caval grafts, the external diameters were successfully reduced by a method of plication devised by one of us (E. J. S.). Plication refers to the infolding of a longitudinal segment of an oversized vein graft by using a continuous Lembert type of suture of 00000 black silk. This infolding of the vein wall results in a reduction of the diameter. Ideally, the Lembert sutures used for this purpose should pass through the adventitia and outer portion of the media without penetration of the intima. This ideal is almost impossible to attain in thin-walled veins so that there is usually more suture material exposed to the blood stream in such altered venous grafts than in the nonplicated venous grafts.

Our results with plicated inferior vena caval grafts are given in Table V. The tabulated grafts are all over 5 cm. in length and showed significant positive graft-aortic disproportion before plication. The reduction of an oversized venous graft diameter by plication definitely eliminates the visible turbulent blood flow in the graft. One graft became occluded by delayed thrombosis after five and one-half weeks, but no early thromboses were observed in the four plicated grafts in mature dogs (0% incidence of early thrombosis as compared to 77.7% in the nonplicated grafts of similar length). This reduction in incidence of early thrombosis occurred in the plicated grafts even though there was a greater amount of suture material exposed to the blood than is usual in our

TABLE V. A COMPARISON OF THROMBOSIS IN PLICATED VENA CAVAL AUTOGRAFTS AND NONPLICATED VENA CAVAL AUTOGRAFTS LONGER THAN 5.0 CM. IMPLANTED INTO THE DOG ABDOMINAL AORTA

TYPE OF GRAFT	NO. OF GRAFTS	AVERAGE DIAMETER BEFORE PLICATION	AVERAGE FER CENT LARGER THAN AORTA	AVERAGE DIAMETER AFTER PLICATION	AVERAGE PER CENT LARGER THAN AORTA AFTER PLICATION	NO. OF EARLY THROMBOSES	PER CENT OF EARLY THROMBOSIS	NO. OF LATE THROMBOSES	PER CENT OF LATE THROMBOSIS
Nonplicated inferior vena caval	9	1.19 cm.	57	----	----	7	77.7	0	0
Plicated inferior vena caval	4	1.29 cm.	68	0.86 cm.	12	0	0	1	25

venous grafts. Thus, the hemodynamics of a venous graft can be improved but another factor which may cause intravascular clotting is introduced.

The data on the late changes in fresh venous autografts to the abdominal aorta of adult dogs were obtained largely by retrograde aortograms at varying periods of time from the time of graft implantation. Aortography was performed under intravenous Nembutal anesthesia by inserting a small caliber polyethylene tube through an exposed femoral artery into the abdominal aorta and injecting 20 c.c. of 70 per cent Diodrast while the x-ray exposure was made.

In some dogs, the graft was exposed by surgical exploration or the graft specimen and adjacent aorta were removed at sacrifice. Graft specimens from sacrificed animals were re-examined after inflation with air at a pressure of 120 mm. Hg. All twenty-two dogs surviving beyond the early postoperative period had at least one aortographic or direct examination of the venous graft eleven days to eighteen weeks after implantation (average, nine weeks). Thirteen dogs had a second aortogram or direct measurement of the graft thirteen to twenty-five weeks after anastomosis of the graft (average, eighteen weeks). One dog (R-86) had a third examination of the graft thirty-three weeks after implantation.

TABLE VI. THE INCIDENCE OF LATE COMPLICATIONS IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN ADULT DOGS ELEVEN DAYS TO THIRTY-THREE WEEKS AFTER IMPLANATION

Total nnmber of dogs surviving 11 days or more	22
Deaths due to late graft complications	0
Delayed thrombosis of the graft	1
Enlargement of graft diameter	22
Diminution in graft diameter	0

One dog (C-61) was found to have a nonocclusive late thrombosis by means of aortography five and one-half weeks after vein grafting the abdominal aorta. The occlusion was complete on aortography ten weeks later and confirmed immediately thereafter at sacrifice of the animal. Two dogs died of superior mesenteric artery thrombosis two to four days after having aortograms. Two dogs were sacrificed to obtain the graft specimens and one other dog (CT) died while under anesthesia on the eleventh postoperative day as he was being explored for thrombosis of the venous graft. The graft was occluded by blood

clot but it was possible to obtain graft measurements. Sixteen dogs are still alive and will be observed over more prolonged periods to ascertain any additional changes in the venous autografts. The late complications seen in fresh venous autografts implanted in the abdominal aorta of mature animals are given in Table VI.

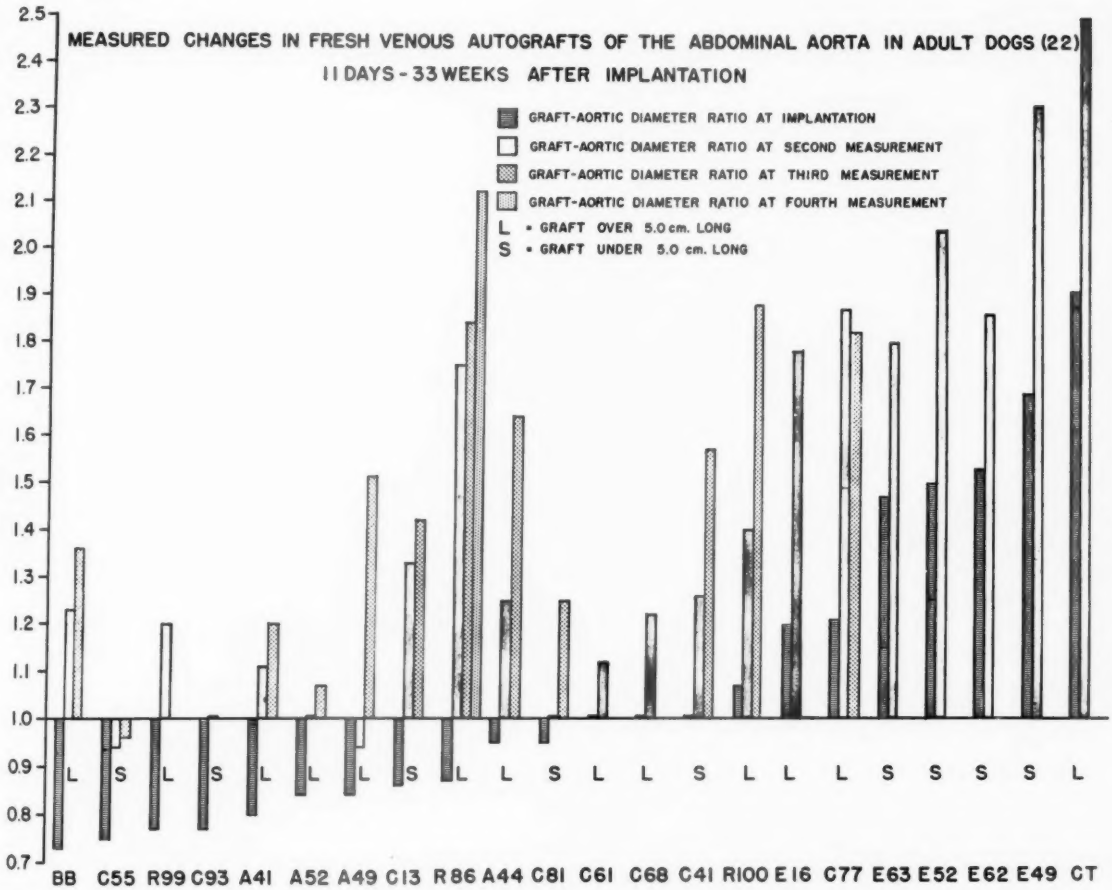

MEASURED CHANGES IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN ADULT DOGS (22)
11 DAYS - 33 WEEKS AFTER IMPLANTATION
GRAFT-AORTIC DIAMETER RATIO AT IMPLANTATION
GRAFT-AORTIC DIAMETER RATIO AT SECOND MEASUREMENT
GRAFT-AORTIC DIAMETER RATIO AT THIRD MEASUREMENT
GRAFT-AORTIC DIAMETER RATIO AT FOURTH MEASUREMENT
L = GRAFT OVER 5.0 cm. LONG
S = GRAFT UNDER 5.0 cm. LONG
2.5
2.4
2.3
2.2
2.1
2.0
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1.0
0.9
0.8
0.7
L S L S L L L S L L S L L S L L L S S S S L
BB C55 R99 C93 A41 A52 A49 C13 R86 A44 C81 C61 C68 C41 R100 E16 C77 E63 E52 E62 E49 CT

Fig. 2.—The measured changes in fresh venous autografts of the abdominal aorta in mature dogs.

Since direct comparison of the actual measurements of the venous grafts and adjacent aorta to the measurement of the Roentgen silhouette of the graft and aorta in the aortograms is not possible, we have made the comparison on the basis of graft-aortic ratios which are valid and comparable. The actual dimensional change in the graft itself is of lesser importance in hemodynamic considerations than is the change in the relative size of the graft and aortic diameters. We are aware that measurements of the x-ray graft-aortic shadows represent the internal diameters of the channels, but in comparing these to the external diameters (in terms of ratios) taken at implantation, we tend to err on the conservative side in the evaluation of the extent of enlargement in the grafts. The graph in Fig. 2 is arranged according to the graft-aortic diameter ratio found in each dog at the time of the vascular anastomosis. Those grafts with biterminal negative disproportion are placed to the left, those grafts with

biterminal positive disproportion are placed to the right, while the central area is occupied by grafts of isodimensional or near-isodimensional proportions. Subsequent changes in graft-aortic ratios in the respective animals are plotted and reveal that enlargement is present in all of the venous autografts which were measured eleven days or more after implantation. Such enlargement was seen as early as eleven days after anastomosis of the graft. It appears that this enlargement occurs irrespective of the relative size of the graft and aorta at the time of implantation. Furthermore, the graphic representations of graft-aortic ratios obtained from the measurement of serial aortograms in some of the animals (Fig. 2) indicate a progressive enlargement of these grafts within the time limits of our observations. Representative examples of the late dimensional changes in grafts with biterminal negative disproportion, isodimensional proportions, and with biterminal positive disproportion are as follows:

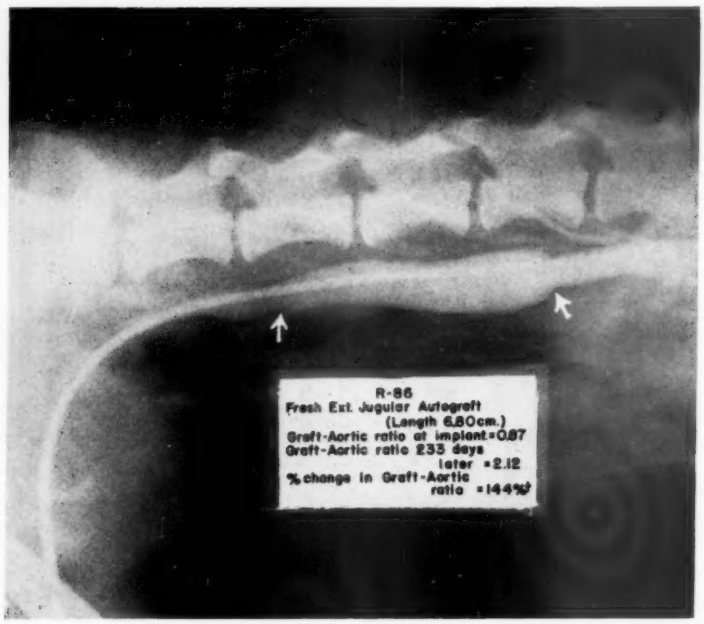


Fig. 3.—Aortogram of dog R-86 taken thirty-three weeks after implantation of a fresh autogenous vein graft into the abdominal aorta. The arrows indicate the lower (left) and the upper (right) graft anastomosis.

Dog R-86 received a fresh external jugular vein graft which was 13 per cent smaller than his abdominal aorta (G/A = 0.87). Fifteen weeks later the graft was exposed at operation and found to be 75 per cent larger than his aorta (G/A = 1.75). An aortogram taken seven weeks after the surgical exploration showed the graft had increased an additional 9 per cent (G/A = 1.84) while the most recent aortogram taken thirty-three weeks after implantation (Fig. 3) revealed the graft was 112 per cent larger than the recipient aorta (G/A = 2.12). This dog is alive and under continued observation.

Dog C-41 had an isodimensional fresh venous autograft implanted in his abdominal aorta. Sixteen weeks later an aortogram showed this graft was 26 per cent larger than the aorta (G/A = 1.26). A second aortogram (Fig. 4)

showed a graft which was 57 per cent larger than the aorta (G/A = 1.57) approximately twenty weeks after being anastomosed into the abdominal aorta. This animal is also under further observation.

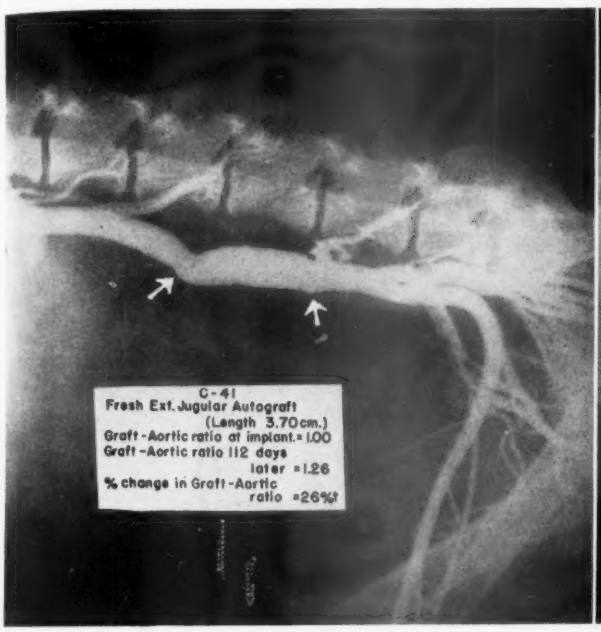


Fig. 4.

Fig. 5.

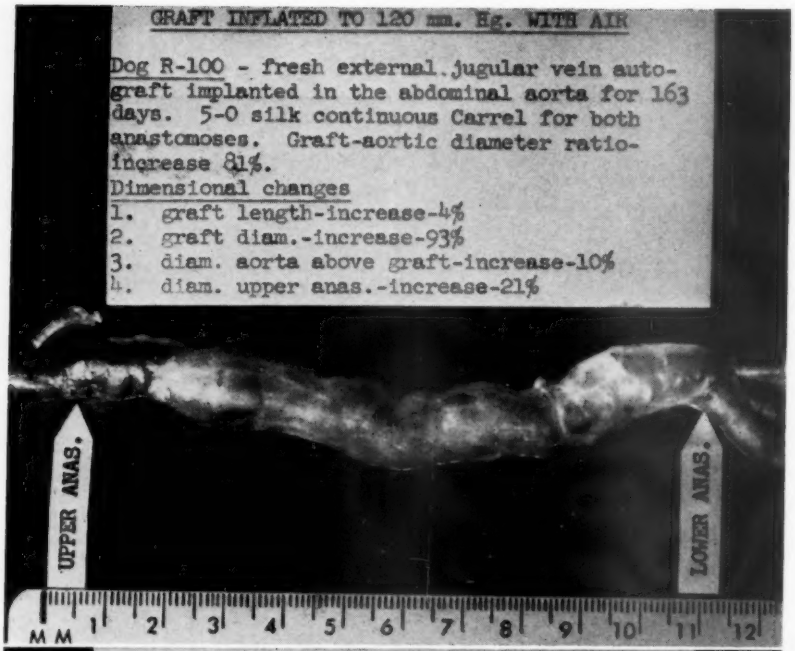
Fig. 4.—Aortogram of dog C-41 taken approximately 20 weeks after implantation of a fresh venous autograft into the abdominal aorta. The arrows point to the upper (left) and lower (right) anastomosis.

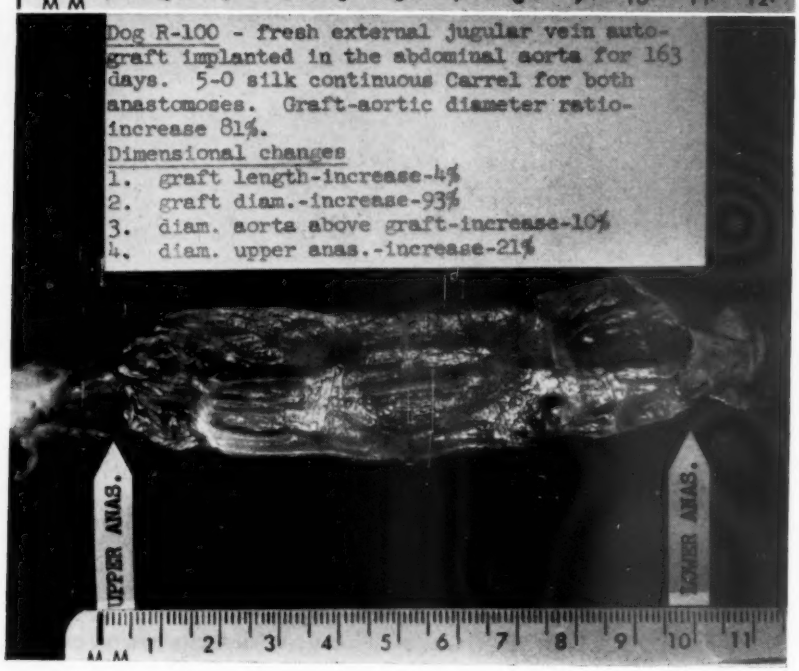
Fig. 5.—Aortogram of dog R-100 taken twenty-three weeks after implantation of a fresh venous autograft into the abdominal aorta. This dog had a thrombosed vena caval graft removed just before the implantation of the graft seen above. The graft is between the white arrows. (Upper anastomosis [left] and lower [right].)

Dog R-100 had a venous autograft which was 7 per cent larger than the recipient aorta (G/A = 1.07). Fourteen weeks later this graft was found to be 40 per cent larger than the aorta (G/A = 1.40) and tortuous in appearance. Twenty-three weeks after implantation of this graft, a second aortogram was made (Fig. 5) and the animal was explored with direct measurements made of the graft and aorta. Immediately thereafter the animal was sacrificed by removal of the graft and adjacent portions of the aorta. The tortuous graft and the aorta were inflated with air under a pressure of 120 mm. Hg (Fig. 6). In general, the graft wall was much thicker than at implantation but some of the dilated areas had a definitely thin wall. The graft-aortic ratios calculated from the aortographic shadow measurements, the graft-aortic diameter measurements in the animal at terminal exploration, and the graft-aortic diameter measurements of the air-inflated specimen are in complete agreement. An open view of the specimen (Fig. 7) showed a smooth endothelial lining throughout with excellent healing at each anastomosis.

II. *Fresh Venous Autografts Implanted in the Abdominal Aorta of growing Pigs.*—Forty-four healthy weanling pigs with an average weight of 13.2

Fig. 6.


GRAFT INFLATED TO 120 mm. Hg. WITH AIR
Dog R-100 - fresh external jugular vein autograft implanted in the abdominal aorta for 163 days. 5-0 silk continuous Carrel for both anastomoses. Graft-aortic diameter ratio-increase 81%.
Dimensional changes
1. graft length-increase-4%
2. graft diam.-increase-93%
3. diam. aorta above graft-increase-10%
4. diam. upper anas.-increase-21%
UPPER ANAS.
LOWER ANAS.
MM 1 2 3 4 5 6 7 8 9 10 11 12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4. diam. upper anas.-increase-21%
UPPER ANAS.
LOWER ANAS.
MM 1 2 3 4 5 6 7 8 9 10 11

Fig. 7.

Fig. 6.—Venous autograft of dog R-100 and adjacent aorta removed after sacrifice of animal and inflated with air under 120 mm. Hg pressure. This is a ventral view of the specimen.

Fig. 7.—Opened view of venous autograft and adjacent aorta of dog R-100 twenty-three weeks after implantation.

kilograms (range 8.3 to 16.9 kilograms) were anesthesized by intraperitoneal Nembutal and supplemental open-drop ether. The abdominal aorta in each animal was exposed by an extraperitoneal approach through a T-shaped flank incision. Strict aseptic precautions were maintained in all operative procedures. The segment of aorta excised in growing pigs prior to graft anastomosis involved the same ratio (1:1.5) as in mature animals. The fresh venous autografts used included eight iliac veins, one renal vein, and the remainder were inferior vena caval segments, fifteen of which were altered by our method of plication. All anastomoses were performed with a continuous Carrel-type suture of 00000 black silk on atraumatic needles. Upon completion of the anastomosis and removal of the Potts clamps, caliper measurements were taken of the graft length, distance from the upper anastomosis to a renal artery, distance from the lower anastomosis to the trifurcation, diameter of the graft, diameter of the aorta midway between a renal artery and the proximal anastomosis, diameters of both anastomoses, and diameter of the aorta below the distal anastomosis. Precautions were used to avoid desiccation of the venous grafts during implantation. Each pig was given 200,000 units of penicillin for two or three days after operation and was kept in a heated pigpen in the laboratories for two weeks. Any vascular complications during this period are listed as early complications. Pigs surviving beyond two weeks were sent to a farm during their period of maturation. Two immediate postoperative pig deaths (cardiac arrest and excess anesthesia, respectively) are eliminated from further consideration, the remaining forty-two animals being considered as providing valid data for the discussion below.

The incidence of early complications is given in Table VII. It was not possible for us to palpate a femoral pulse in the young pig but the pigs with venous grafts of the abdominal aorta which demonstrated motor incoordination of the hind limbs after operation were considered by us as having thrombosed grafts. A series of nine pigs with venous grafts but without evidence of motor incoordination on exercise were explored one to ten days following graft implantations and all of these grafts were found open to inspection and palpation. One of the three grafts which thrombosed early was poorly plicated on two attempts and probably should be eliminated as a technical failure. Rupture of a graft occurred once in each of the three principal categories of grafts: with isodimensional proportions, with biterminal positive disproportion, and with biterminal negative disproportion. One of these ruptured grafts was plicated and the longest (5.3 cm.) in this series of pigs. The average length of the other vein grafts was 2.29 cm. (range 0.9 to 4.2 cm.). One pig died on the fourth

TABLE VII. THE INCIDENCE OF EARLY COMPLICATIONS IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN GROWING PIGS

Total number of fresh venous autografts	42
Deaths due to early graft complications	6* (14.2%)
Early thrombosis of grafts	3 (7.2%)
Anastomosis dehiscence	0
Ruptured graft	3 (7.2%)
Number of grafts observed beyond two weeks	35 (83.2%)

*One additional death not related to graft.

postoperative day from an unknown cause, but the vein graft was found open at autopsy.

A total of thirty-five growing pigs survived beyond the early postoperative period. All of these pigs eventually had their grafts removed for examination after slaughter or autopsy. The late results in this group are listed in Table VIII. The pigs which came to slaughter twenty-two to thirty weeks after graft implantation increased their weight six- to eightfold. Occluded grafts were thin fibrous cords and were not further examined. There were twenty-three grafts with delayed thrombosis, but included among these are three plicated grafts which were definitely technical failures. The twenty valid venous grafts, plicated and nonplicated, with delayed thrombosis had an average diameter at implantation of 0.60 cm. (range 0.30 to 1.20 cm.) while the recipient aorta average diameter was 0.55 cm. (range 0.45 to 0.75 cm.). In the ten satisfactorily plicated vein grafts subject to late complications, six (60 per cent) had delayed thrombosis. Eight of the nine grafts which were open at surgical exploration in the early postoperative period were found to be occluded and cordlike in appearance three to thirty weeks later.

TABLE VIII. LATE CHANGES OBSERVED IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN GROWING PIGS THREE TO THIRTY WEEKS AFTER IMPLANTATION

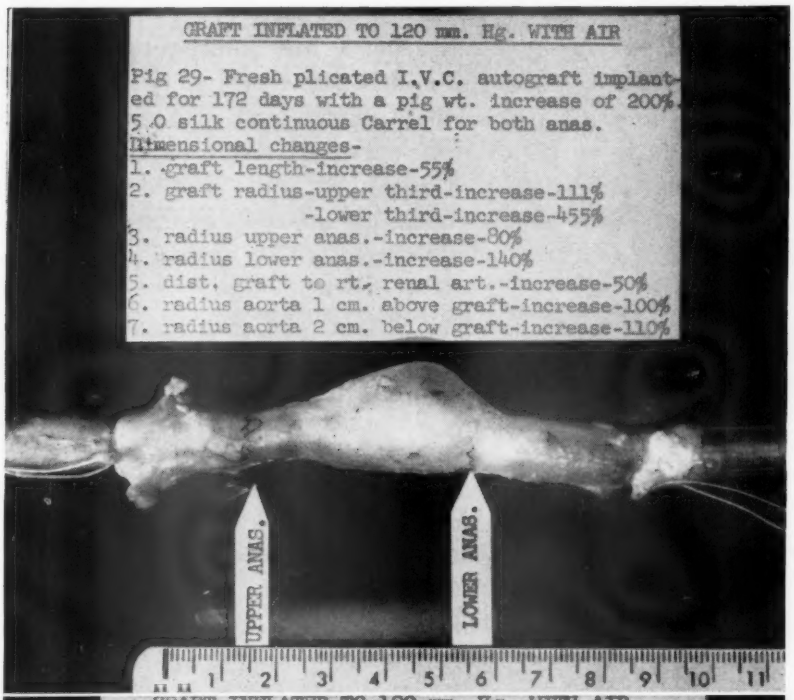
Total number of pigs surviving beyond two weeks	35
Delayed thrombosis of the graft	20 (57%)
Aneurysmal enlargement of graft	7 (20%)
Relative decrease in graft diameter	5 (14%)
Delayed thrombosis due to technical errors in plication	3 (9%)

The relationship of graft-aortic diameter ratios at implantation to the incidence of late thrombosis in fresh venous autografts is given in Table IX. Under the conditions of our experiments, the differences in the percentages of delayed thrombosis in the near-isodimensional grafts, negative disproportionate grafts, and positive disproportionate grafts are not significant because of complicating factors. These factors will be discussed later.

Twelve fresh venous autografts of the abdominal aorta in growing pigs were found open at slaughter (34 per cent of the pigs showing no early complications). At implantation the open grafts had an average diameter of 0.64 cm. (range 0.40 to 0.85 cm.) while the host aorta diameters averaged 0.49 cm. (range 0.35 to 0.55 cm.). After being freed of excess connective tissue, these graft specimens were distended by air at a pressure of 120 mm. Hg and measurements taken as previously outlined. The analysis of these measurements is recorded in Table X. Seven of these open grafts showed a ventral or lateral aneurysmal dilatation (Fig. 8). The remaining five grafts had no dilatations, but showed a relative decrease in graft diameter inasmuch as the host aorta enlarged to a greater degree with growth than the venous graft up to the time of our observations (Fig. 9).

A true representation of the dimensional changes which occurred in the grafts with aneurysmal dilatation was not possible by the comparison of graft-aortic diameter ratios at implantation to the graft-aortic *diameter* ratios found at later observations. An increase in diameter does not adequately portray the seriousness of an eccentric aneurysmal dilatation. A valid and meaningful

Fig. 8.


GRAFT INFLATED TO 120 mm. Hg. WITH AIR
Pig 29- Fresh plicated I.V.C. autograft implanted for 172 days with a pig wt. increase of 200%. 5 0 silk continuous Carrel for both anas.
Dimensional changes-
1. graft length-increase-55%
2. graft radius-upper third-increase-111%
-lower third-increase-455%
3. radius upper anas.-increase-80%
4. radius lower anas.-increase-140%
5. dist. graft to rt. renal art.-increase-50%
6. radius aorta 1 cm. above graft-increase-100%
7. radius aorta 2 cm. below graft-increase-110%
UPPER ANAS.
LOWER ANAS.

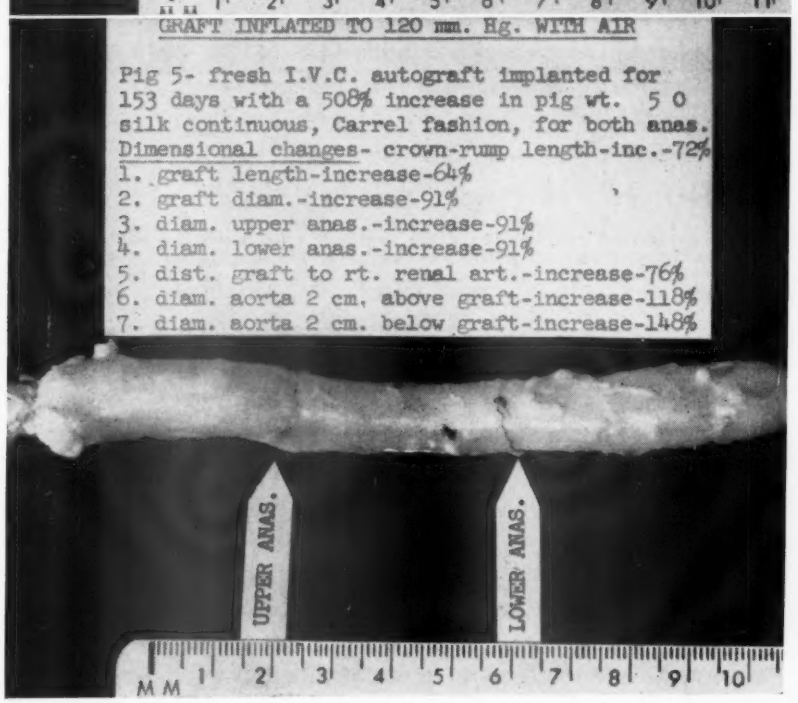

GRAFT INFLATED TO 120 mm. Hg. WITH AIR
Pig 5- fresh I.V.C. autograft implanted for 153 days with a 508% increase in pig wt. 5 0 silk continuous, Carrel fashion, for both anas.
Dimensional changes- crown-rump length-inc.-72%
1. graft length-increase-64%
2. graft diam.-increase-91%
3. diam. upper anas.-increase-91%
4. diam. lower anas.-increase-91%
5. dist. graft to rt. renal art.-increase-76%
6. diam. aorta 2 cm. above graft-increase-118%
7. diam. aorta 2 cm. below graft-increase-148%
UPPER ANAS.
LOWER ANAS.

Fig. 9.

Fig. 8.—Pig 29. Lateral view of venous autograft and adjacent aorta inflated with air under 120 mm. Hg. Note how the force of the arterial blood pressure was probably directed against the wall of the aneurysmal dilatation.

Fig. 9.—Pig 5. Ventral view of venous autograft and adjacent aorta inflated with air under 120 mm. Hg. The graft is slightly smaller than the aorta since it failed to grow as rapidly.

expression of the data on graft dilatation was made by using the aortic radius measurements and the "maximum eccentric graft radius" (the "maximum eccentric graft radius" is measured from a line joining the midpoint of the upper and lower anastomoses to the most distant periphery) measurements for the determination of graft-aortic ratios before and after implantation. Fig. 10 shows the results in terms of graft-aortic diameter ratios as well as graft-aortic radius ratios. As in the mature animals, the dilatation which occurred in fresh venous autografts of the abdominal aorta in growing pigs was not related to the nature of the graft-aortic diameter (or radius) disproportion present at implantation.

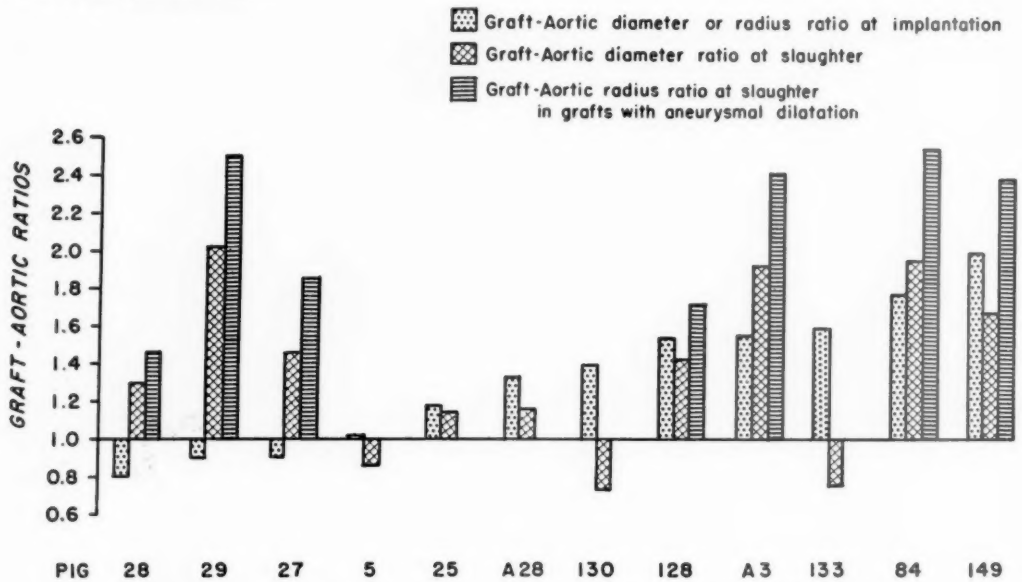


Fig. 10.—The measured changes in fresh venous autografts of the abdominal aorta in growing pigs patent twenty-two to thirty weeks after implantation. A comparison of these changes when expressed from diameter measurements and from radius measurements.

DISCUSSION

The study of the use of venous autografts in the repair of arterial defects is not new. Our series of investigations differs from others by the greater number of venous grafts observed, and the evaluation of graft-length and diameter-disproportion factors in the development of early and late graft complications.

In the mature animals, long fresh venous autografts and short fresh venous autografts of the abdominal aorta with comparable degrees of biterminal positive disproportion showed dissimilar tendencies toward intravascular clotting (Table II). The reason for this difference in the frequency of early thrombosis in the two groups appears to be related to *duration* of turbulent blood flow (Fig. 1). The long grafts are exposed to more prolonged turbulent flow causing repeated contacts between the cellular blood components and intimal surfaces (Fig. 1). When this more prolonged turbulent flow is combined with a concomitant decrease in the velocity of blood flow over a longer venous graft segment, the incidence of thrombosis is great. In the short-length grafts with biterminal positive disproportion, the same combination of circumstances exists, but the duration of abnormal flow is brief and early thrombosis is not as likely

to occur. This belief is supported by our data on venous grafts in mature and growing animals. In the growing pigs, all the fresh venous autografts to the abdominal aorta (except one) were of short length and the incidence of early thrombosis in these grafts was low.

The zonal division of the blood with normal flow (Fig. 1) is pertinent to a possible explanation of the occurrence of delayed thrombosis. In vessels with larger intraluminal dimensions, as in the adult dogs, the depth of the peripheral plasma zone is greater than in the small-sized vessels found in young pigs. Where the clear peripheral zone is of greater magnitude, the exposed suture material or the fibrin seal of the needle holes and graft-aortic juncture are less likely to protrude into the axial core of moving cellular elements of the blood. In twenty-two dogs with venous grafts subject to late thrombosis, the average graft diameter at implantation was 0.88 cm. and the recipient aorta average diameter was 0.72 cm. One of these grafts showed delayed thrombosis. This graft had its diameter reduced by plication. In vessels of smaller diameters, the clear zone is of shallow depth and suture material or the fibrin seal may easily extend into the axial cellular zone with resultant small areas of turbulent flow and eventual clot formation. Early thrombosis does not occur frequently under these circumstances because the degree of turbulence is very slight and thrombus formation is probably by a slow process of accretion. At present, the cause of the delayed thrombosis in smaller grafts is not proved. Studies now in progress in our laboratories may reveal histologic changes in the walls of venous grafts which will explain the occurrence of late thrombosis in grafts of small diameter. In the thirty-five growing pigs in which the venous grafts were examined in the late postoperative period, the average graft diameter at implantation was 0.62 cm. and the recipient aorta average diameter was 0.52 cm. These are external diameter averages so that the intraluminal cross-sectional areas of young pig vessels are considerably smaller than in vessels of adult dogs. Twenty growing pigs showed delayed thrombosis of the venous autografts implanted into their abdominal aortas.

TABLE IX. THE DISTRIBUTION OF DELAYED THROMBOSIS IN FRESH VENOUS AUTOGRAFTS OF THE ABDOMINAL AORTA IN GROWING PIGS AS RELATED TO GRAFT-AORTIC DIAMETER RATIOS AT IMPLANTATION

COMPARED TO AORTA AT IMPLANTATION	NO. OF GRAFTS	DELAYED THROMBOSIS
Grafts 21 to 48% smaller	6	5 (83%)
Grafts 20% smaller to 20% larger	11	7 (64%)
Grafts 21 to 100% larger	15	8 (53%)
Total	32*	20

*Three animals eliminated on the basis of technical errors.

The incidence of delayed thrombosis in growing pigs (Table IX) is slightly greater in the group of venous grafts with more satisfactory hemodynamic qualities (20 per cent smaller to 20 per cent larger than the recipient aorta) than in the group of grafts with one type of undesirable hemodynamic qualities (21 per cent to 100 per cent larger than the aorta). We believe the significant point of difference in these two groups is the amount of suture material placed within the lumen of the grafts. None of the oversized grafts were plicated,

whereas a large majority of the near-isodimensional group consisted of plicated grafts. The high percentage of delayed occlusion in grafts 21 per cent to 48 per cent smaller than the host aorta is believed to be related to the very small diameters of these transplants (average diameter 0.37 cm.).

Positive disproportion in a venous graft can be reduced or eliminated by longitudinal plication of the wall. Grafts altered by this method show no visible turbulence in the blood flow. However, such displacement of poor hemodynamics is done at the expense of introducing another factor which may result in thrombosis within the graft. Furthermore, technical difficulties such as irregular dimensions after plication or necessity of revising the plication suture may greatly endanger the successful function of a plicated venous graft.

Stenosis of a graft or constriction at the site of anastomosis did not occur in any of the fresh venous autografts in our studies. Some degree of enlargement occurred in all the venous autografts we studied. This finding of venous graft enlargement is in agreement with the reports of Shumacker and associates (1950), Sako (1951), Sauvage and Harkins (1952), and Johnson and associates (1949 and 1951). All of those investigators reported grafts of short length while our series included grafts of longer length. Johnson and associates (1949) reported the use of seventeen venous grafts measuring from 1.0 to 4.6 cm. in length. They believed there was dilatation of "longer grafts" but not in the "shorter grafts." Our data on venous grafts fail to show any correlation between subsequent enlargement of the grafts and their respective lengths at implantation. Taylor (1951) stated ". . . if a vein of approximately the same diameter as the artery was selected, then no dilatation occurred." This statement finds no support in the later diameter changes we observed in venous grafts which had isodimensional proportions at implantation. Although Anzola, Palmer, and Welch (1952) reported no dilations in venous autografts implanted into femoral arteries, the absence of dilatation of those grafts may be related to the supporting effect of the adjacent musculo-fascial structures. The abdominal and thoracic aorta are not surrounded by muscles or strong fascia so that any tendency toward graft enlargement implanted in those large vessels is relatively uninhibited. We have a series of dogs under observation with venous and arterial transplants to the abdominal aorta, thoracic aorta, and femoral artery. The information to be obtained from those observations may clarify the role of surrounding and supporting tissues in the dimensional changes of vascular grafts.

Eccentric aneurysmal dilatations of venous grafts in our mature dogs may be present but could not be positively identified since it was not possible to make more than one satisfactory x-ray exposure during the intra-aortic Diodrast injection. In the growing pigs, eccentric aneurysmal dilatation of the patent venous grafts was frequently found (Table X and Fig. 8). It seems likely that such aneurysmal dilatations are more undesirable than the fusiform types and deserve special emphasis. It is significant to us that all of these aneurysmal dilatations occurred on the lateral or ventral aspects of the grafts. No dilatations were found on the posterior aspects where the grafts lie in close apposition to the psoas muscles. Johnson and associates (1951) described a "saccular aneurysm" in a venous autograft implanted into the abdominal aorta of a

growing pig, but did not record the direction of this dilatation. Whether such aneurysmal dilatations of venous grafts of the abdominal aorta will progress to eventual rupture is not known at present. Johnson (1952) reported rupture of two venous autografts of the thoracic aorta in growing pigs. In a series of unpublished experiments, we have observed rupture of a greatly dilated venous autograft of a young pig's thoracic aorta forty-four days after implantation. It is worth emphasizing that these ruptures developed in venous grafts which were unsupported by any adjacent muscles or fascia.

TABLE X. THE RESULTS IN TWELVE FRESH VENOUS AUTOGRAFTS FOUND PATENT AT SLAUGHTER IN GROWING PIGS TWENTY-TWO TO THIRTY WEEKS AFTER GRAFT IMPLANTATION

DESCRIPTION OF GRAFT	NUMBER	PER CENT
Grafts showing no dilatation	5	42
Grafts showing aneurysmal dilatation	7	58

Thirteen dogs had two or more aortograms from two to thirty-three weeks after implantation of a venous graft into their abdominal aortas. One dog was found to have delayed thrombosis. Another dog failed to show any further enlargement of his venous graft in the time between the first and second aortograms (ten weeks) although the graft-aortic diameter ratio increased 54 per cent from the time of implantation to the first aortogram. In the other eleven dogs there was a progressive increase in their graft-aortic ratios in serial aortograms, indicating a continued enlargement in the graft diameters up to the time of our observations (thirteen to thirty-three weeks). Sako (1951) observed an initial dilatation of venous autografts of the thoracic aorta in dogs, but no further enlargement occurred after six months. A sufficient number of venous autografts has not been studied over long periods to establish their ultimate dimensional fate. Dilatation of a venous graft implanted into a popliteal artery was seen by Freeman (1949) three weeks after operation, but after fifteen months this graft showed a decrease in its diameter to isodimensional proportions. The change in the size of this graft may be related to the supporting effect of the adjacent muscular structures. Sixteen of our dogs with venous grafts of the abdominal aorta are still alive and will form the basis for our long-term observations of grafts without adjacent supportive tissues.

Five venous autografts transplanted into the abdominal aorta of growing pigs demonstrated a decrease in the graft-aortic ratios from the time of implantation to slaughter approximately six months later. It appears that under the conditions of our experiments, some venous autografts fail to keep pace with the rapid growth of the aorta in growing pigs. None of these grafts could be classified as stenosed. This failure of sufficient enlargement was seen in grafts which were the same diameter or larger than the aorta at operation. Everson and Southwick (1951) and Johnson and associates (1951) found equivalent degrees of growth in auto-vein grafts and the host aortas of growing pigs.

SUMMARY

1. Forty-two valid fresh venous autografts in growing pigs and thirty-six valid fresh venous autografts in mature dogs form the basis for this report. All of these grafts were implanted into the abdominal aortas of the animals.

2. Fresh venous autografts served as satisfactory conduits for blood, provided occlusion by thrombosis did not occur.

3. The factors of graft length and biterminal positive disproportion were highly significant in the development of early thrombosis.

4. Short-length fresh venous autografts with positive disproportion, negative disproportion, or made isodimensional by plication and implanted into the abdominal aorta of growing pigs had a high incidence of delayed thrombosis.

5. Autogenous vein grafts which remained patent had a tendency to dilate. This tendency was not related to the length of the grafts nor to the graft-aortic diameter ratio at the time of implantation.

6. Aneurysmal dilatations were found in a significant number of venous autografts in growing pigs which were found open six months after implantation. However, a lesser number of autoplastic vein grafts did not grow as rapidly as the host aorta.

7. Observations are being recorded on fresh venous autografts of the abdominal aorta in mature dogs implanted longer than thirty-three weeks.

CONCLUSIONS

1. Under the conditions of our experiments, fresh venous autografts with biterminal positive disproportion of 21 per cent or more and longer than 5.0 cm. in length show a very high incidence of early thrombosis after implantation in the abdominal aorta of mature dogs.

2. Fresh venous autografts show a definite tendency toward dilatation when implanted into the abdominal aorta of growing pigs and adult dogs.

3. The graft-aortic diameter ratio at implantation in growing pigs and mature dogs appears not to be related to the subsequent enlargement or aneurysmal dilatation of the fresh venous autograft.

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ANEURYSM FORMATION IN EXPERIMENTAL VEIN GRAFTS IN THE THORACIC AORTA

JULIAN JOHNSON, M.D., CHARLES K. KIRBY, M.D., AND JAMES D. HARDY, M.D., PHILADELPHIA, PA.

(From the Harrison Department of Surgical Research, School of Medicine, University of Pennsylvania, and the Department of Surgery of the University of Pennsylvania and the Children's Hospital of Philadelphia)

ALTHOUGH it has been demonstrated by Gross[1] and others that preserved aorta homografts function satisfactorily for several years at least, there is still some question as to whether the use of such grafts is the best possible solution to the problem of a long defect in coarctation of the aorta. Extensive changes of a degenerative type, including calcification, occur in the graft walls, presumably as the result of tissue incompatibility. It will be many years before we know whether these changes may eventually lead to thrombosis, aneurysm formation, or rupture. We are particularly concerned about the advisability of using aorta homografts in children, since it has been found that these grafts do not increase in size satisfactorily during the growth of experimental animals.[2, 3]

Because of these doubts concerning homografts, there is continued interest in using autogenous tissue to bridge long defects in coarctation of the aorta. Interest has centered on autogenous vein grafts because autogenous artery grafts of adequate size cannot be safely obtained and because autogenous vein grafts have proved satisfactory in smaller arteries in many patients. Some of our experiences with autogenous vein grafts, both experimental and clinical, have been previously described.[4] On the basis of these experiences we have considered the possibility of using jugular vein grafts in patients with coarctation of the aorta when the defect is too long for an end-to-end anastomosis.

In our previous experiments with vena cava grafts to the abdominal aorta in pigs,[5] we found that the grafts did grow, and we attributed some dilatation to the fact that the grafts were larger than the aorta when they were implanted. A real aneurysm occurred in only one instance. It seemed advisable, however, to observe the behavior of vena cava grafts in the thoracic aorta.

In preliminary experiments in dogs, Gerbode[6] found that vein grafts did well in the thoracic aorta. We studied the problem in growing pigs, thinking particularly of the problem in children. Since we had observed previously in the abdominal aorta that short vena cava grafts (less than 1.5 cm.) were much less likely to dilate than longer ones, it was thought best to use fairly long grafts (1.5 to 3.5 cm.). It was decided to use vein grafts no greater in diameter

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than the recipient aorta in order properly to evaluate dilatation. The problem of whether the grafts might be strengthened by covering them with pericardium was considered.

EXPERIMENTAL METHOD

Pigs which weighed about 25 pounds were used in the experiments. A segment of the vena cava just below the renal vein was removed through a right flank incision. After closing this incision, the left chest was opened through a fourth interspace incision. A segment of the aorta in the coaretation area, somewhat shorter than the vein graft to be used, was excised and the vena cava graft was sutured into place. Sutures of 00000 Deknatel silk were interrupted at several points to prevent constriction at the suture line.[7] A glass cannula was used to connect the aortic ends during the suturing, and aortic blood flow was never interrupted for more than five minutes at a time. The cannula was removed after about two-thirds of the second suture line was completed. In six of the twelve animals operated upon, the vena cava graft was wrapped with a piece of the pig's own pericardium. The edges of the pericardium were sutured together. The ends extended beyond the two suture lines.

In no instance was the vein graft larger in diameter than the recipient aorta. The length of the vein graft as measured in situ varied from 1.5 cm. to 3.2 cm. Only two were less than 2.6 cm. The edges of the mediastinal pleura were carefully approximated over the graft.

On the first postoperative day the animals were taken to the university farm, where they were raised. All surviving animals were sacrificed when they had attained a weight of about 250 pounds, an eight- to tenfold increase. The grafts were excised together with short aortic segments above and below. Measurements and photographs were taken with the specimens inflated to a pressure of 120 mm. Hg.

RESULTS

In the group without a pericardial covering, five animals survived. One graft was contracted, with a lumen about 1.0 mm. in diameter. It had apparently undergone thrombosis soon after insertion. The other four grafts were greatly dilated and two of these showed definite aneurysm formation in addition to generalized dilatation. One of the specimens is shown in Fig. 1.

In the group with a pericardial covering, only two animals survived long enough for significant observations to be made. One died of hemorrhage a short time before it was to be sacrificed. A large perforation was found in the graft wall (Fig. 2). There was no evidence of infection in the region of the graft. The other graft had undergone moderate dilatation despite the pericardial covering (Fig. 3). The pericardium appeared to be only slightly thickened and was easily peeled away from the surface of the vein graft.

DISCUSSION

The experimental series which we have described is not large, and the results obtained are not necessarily those which might be found in patients. The high incidence of extensive dilatation and aneurysm formation cannot be

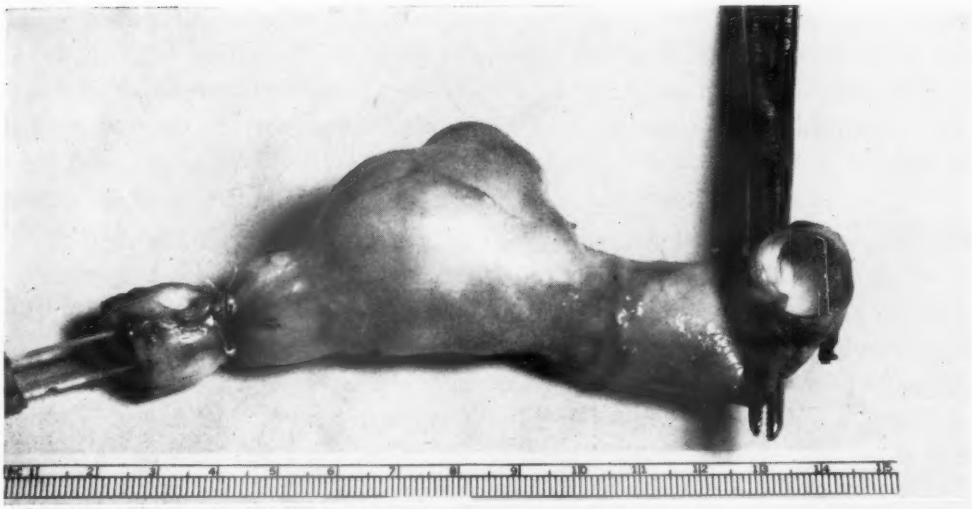

Fig. 1.

Fig. 2.

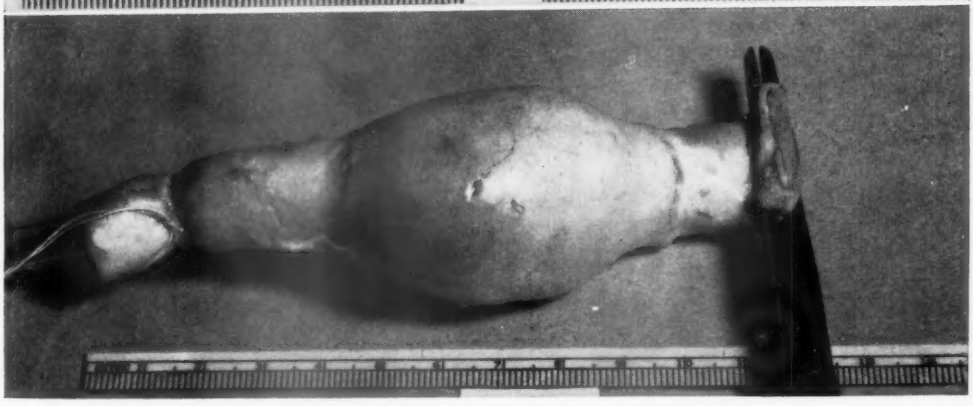

Fig. 3.

Fig. 1.—A vena cava graft to the thoracic aorta, without pericardial covering. A definite aneurysm has formed. The diameter of this graft was 2 mm. less than that of the aorta when it was inserted.

Fig. 2.—A vena cava graft to the thoracic aorta, covered with pericardium. Spontaneous perforation of the graft wall occurred shortly before the animal was to be sacrificed. The suture lines were intact. This specimen is somewhat distorted because it was placed in fixative solution before it was photographed.

Fig. 3.—A vena cava graft to the thoracic aorta, covered with pericardium. There has been considerable dilatation.

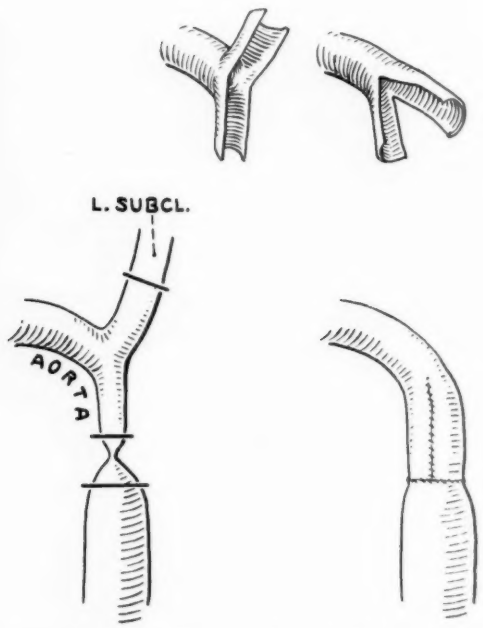

Fig. 4.—Illustration of a method used in one 8-year-old patient to overcome a long defect in coarctation of the aorta. The aorta between the subclavian and the coarctation, and the subclavian artery were used to create one vessel of adequate lumen.

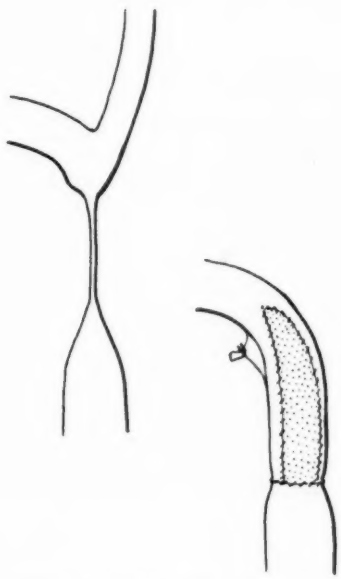

Fig. 5.—Illustration of a method which may be feasible in children for increasing the diameter of the subclavian artery when better methods for overcoming the defect cannot be used. The arterial patch may be secured from the radial, ulnar, or possibly the internal mammary artery.

lightly dismissed, however. We, at least, are convinced that vein grafts should not be used in the thoracic aorta clinically unless it can be shown in larger experimental series that the incidence of complications is much lower than now seems apparent. Strengthening the graft by covering it with additional autogenous tissue may eventually prove to be helpful. Our experience to date with the use of pericardium for this purpose does not seem promising.

If it is granted that vein grafts should not be used to bridge defects in coarctation of the aorta unless more favorable evidence is forthcoming, the best solution for adults, at present, is the use of aorta homografts. This solution is not applicable to children, since aorta homografts probably do not increase adequately in size during growth. This raises the question as to whether these children should be operated upon. Would it perhaps be better to wait until they have attained full growth before operating upon them in the event that a graft should be needed?

We feel rather strongly that such a policy should not be adopted. The age from about 8 to 12 years seems to us ideal for operation. The vessels are usually still pliable and elastic, and the operation can be performed with greater safety than in adult life. A longer defect may be overcome by end-to-end suture when the aorta is still elastic and certain plastic procedures may be undertaken which might be more hazardous in the older age group.

The use of the subclavian artery for anastomosis to the descending aorta has been disappointing in about one-half the instances in which it was used. This is probably because the subclavian in those instances was not large enough. In an 8-year-old girl recently operated upon we found the constriction to be about 3.0 cm. below the origin of the subclavian. The aorta between the subclavian and the coarctation was not adequate in diameter at any point, so that little was to be gained by going higher on it. The subclavian likewise was apparently not large enough to carry the load of the aorta. By means of the plastic procedure illustrated (Fig. 4) we were able to use this portion of the aorta and the subclavian artery to create one vessel with a large lumen. This served the purpose well. The patient's blood pressure returned to normal (110/70) and has remained so for eight months.

When the subclavian artery has to be turned down and is inadequate in size, the aorta between it and the coarctation often cannot be used to enlarge it. In such instances it might be possible to enlarge the subclavian by inserting a patch graft consisting of a segment of the radial, ulnar, or internal mammary artery in the manner illustrated (Fig. 5).

SUMMARY

Vena cava grafts, with and without pericardial covering, were implanted in the thoracic aortas of pigs. There was a high incidence of complications, particularly of dilatation and aneurysm formation. These results suggest that it may be unwise to use vein grafts in the thoracic aorta in patients.

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A CRITICAL STUDY OF PRESENT CRITERIA GOVERNING SELECTION AND USE OF BLOOD VESSEL GRAFTS

RALPH A. DETERLING, JR., M.D., MARY S. PARSHLEY, PH.D., AND J. WALLACE BLUNT, M.D., NEW YORK, N. Y.

(From the Department of Surgery, College of Physicians and Surgeons, Columbia University, and the Surgical Service of Presbyterian Hospital)

ALTHOUGH the promise of clinical applicability of vessel transplants could be derived from the pioneer experimental work of Höpfner,[1] Stich,[2] Carrel,[3-5] Guthrie,[6, 7] and others, the confirming studies by Gross and his coworkers[8-10] and others made the use of vessel grafts in man secure. It was immediately evident that certain criteria for selection of material would have to be established, as well as the recommendation of a method and optimal period of storage of these grafts. On the basis of limited studies and certain assumptions, rather strict limitations were initially imposed which have markedly reduced available material for vessel banks.

The poor results with frozen material in Gross's early experiments contrasted markedly with the success noted with grafts preserved at 4° C. in a balanced electrolyte solution, containing serum and glucose. The fact that cells of the latter grafts could be grown by tissue culture during a month or so of storage in the nutrient medium suggested that viability was an important requisite for functional success. Supporting this concept were the findings of Swan and associates.[11] They indicated that, after implantation, an accelerated disappearance of normal histologic structure occurred if storage of the graft in a nutrient medium had been prolonged beyond 42 days. This coincided with the period of loss of viability. As a result, most vessel banks have employed similar methods of storage, discarding the grafts at 6 weeks.[12]

This waste of graft material and the high cost of maintaining such a bank impressed us with a need for further experiments aimed at determining the true importance of viability in regard to functional success, and at evaluating other methods of preservation if viability were not all important. Our success with certain methods of quick freezing[13-15] confirmed that noted by Hufnagel,[16] and approximated that achieved in our laboratory with grafts stored in a nutrient medium at 4° C.[15] In none of these frozen grafts were viable cellular elements demonstrated by tissue culture. Encouraged by these findings, we have explored the techniques of electronic irradiation[17] and lyophilization* which permit storage of aortic tissue for a very long period of time. The early results have indicated a high degree of functional success. In none

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of these grafts has cellular growth been demonstrated by tissue culture. It would therefore appear that viability is not a critical factor in blood vessel storage.

To test this concept further we implanted into the abdominal aorta of adult dogs 5 aortic homografts which had been stored in a nutrient solution* at 4° C. for periods from 48 days to 212 days in a sterile state (Table I). No cellular growth was demonstrated by tissue culture. These animals were observed for up to two years and, in all, there was complete functional success of the graft. In none of the segments were there gross signs of degeneration greater than that noted in our normal series of nutrient grafts. There was a centrally placed intimal erosion in 2, but calcification was not encountered. Microscopically, the grafts appeared similar to comparable ones which had been stored for less than 42 days. Vessel segments which have been stored for two years in the nutrient medium are being studied at present.

TABLE I. RESULTS OF THE USE OF GRAFTS STORED AT 4° C. IN A NUTRIENT ELECTROLYTE SOLUTION FOR OVER SIX WEEKS

DOG	AGE OF GRAFT (IN DAYS)	TIME IN DOG (IN DAYS)	FUNCTION	THROMBOSIS	EROSION	CALCIFICATION
258	48	62	+	0	+++	0
784	53	52	+	0	0	0
470	82	744	+	0	0	0
38	125	505	+	0	+	0
87	212	100	+	0	0	0

We would not draw conclusions from this small series, but think the results are in accord with our previous observations regarding cellular viability and functional success.[15] Swan and associates[11] also noted satisfactory function in a larger and similar group of dogs with grafts which had been stored for from 6 weeks to 6 months. They thought, as noted before, that the degeneration of the graft after implantation was less rapid or pronounced in other dogs with grafts which had been stored for less than 40 days. It is not our intention to encourage the use of grafts stored for long periods in the nutrient medium. We believe that other methods of preservation under study will permit a great extension of the storage period with far less danger from fungus or bacterial growth.

Perhaps the most limiting factors have been the restrictions set in regard to the acceptability of donor individuals. The upper age has usually been set at 45 years[12] although, according to some, 35 years should be maximum.[18, 19] There is general agreement insofar as exclusion of individuals younger than 2 to 3 years of age. It has been thought that the small diameter of the aorta and occasional friability of such tissue would make such grafts unsatisfactory, except for smaller peripheral vessels. Most surgeons have used fresh autogenous veins in such locations, but there has been a report of the successful use of a stillborn infant's aorta for repair of a popliteal artery after excision of a luetic aneurysm. The authors experienced no technical difficulty with the graft which had been preserved for 4 days.[20] The time interval following

*Simms' X6 solution, containing 10 per cent homologous dog serum, 50 units each of penicillin and streptomycin per cubic milliliter, and .005 per cent phenol red.

death, during which the vessels should be obtained, has been set at 6 hours by many and there are a few who have thought 3 hours to be preferable. To those who have had experience in obtaining donor material, this time limitation provides a formidable barrier.[21] The time required for notification of relatives and responsible personnel is often considerable. The mobilization of the team designated to obtain vessel grafts, as well as the preparations, consumes additional time. It is possible that the rush to meet this deadline has contributed to the loss of material through contamination. It has not been surprising that extension of the number of acceptable causes of death or pertinent medical conditions has been proposed in order to obtain an adequate supply of vessels, in compliance with the above limitations.

In order to determine the advisability of adhering to our present strict criteria, we have analyzed a series of 500 autopsies performed at Presbyterian Hospital in New York between May, 1949, and February, 1951. All cases in which restrictions on the extent of the autopsy would have prevented obtaining vessel grafts have been excluded. Furthermore, children younger than 3 years of age have not been included.

Interval Between Death and Autopsy.—With few exceptions the time of death was reasonably well known. While the necessary personnel and relatives were being informed of the death, preparation of the body was carried out. Transport to the refrigerated cubicles in the morgue occurred in most instances within an hour and a half. On rare occasions, because of visiting hours or other reasons, there was a delay of as long as two hours before the body was refrigerated at 4° C.[22]

Of the 500 cases, autopsy was performed in less than 6 hours in 221 or 44.2 per cent (Table II). This high incidence was surprising to us since no particular effort to speed the procedure was made in this group. Autopsy was begun within 3 hours in 84 or 17 per cent. Most significant was the fact that 482 or 96.4 per cent were performed within 24 hours.

TABLE II. THE TIME INTERVAL BETWEEN DEATH AND AUTOPSY, FROM FIVE HUNDRED CASES AT PRESBYTERIAN HOSPITAL, NEW YORK

HOURS	CASES	PER CENT	
0 to 6	221	44.2	96.4% within 24 hours
6 to 12	140	28.0	
12 to 18	93	18.6	
18 to 24	28	5.6	
24 to 36	10	2.0	
36 to 48	2	.4	
Over 48	6	1.2	
Total	500	100	

Sex and Age Incidence.—There were 293 men and 207 women in this series of 500 autopsies. Only 20 per cent of the patients were below 40 years of age. The greatest incidence was in the sixth decade (24 per cent), and almost one-half of all the patients were in the sixth and seventh decades (Table III).

This preponderance of cases in the middle age could be considered as representative of any comparable hospital with a similar patient population. It

TABLE III. THE AGE AND SEX INCIDENCE FROM FIVE HUNDRED AUTOPSIES PERFORMED AT PRESBYTERIAN HOSPITAL, NEW YORK

AGE (IN YEARS)	MEN	WOMEN	TOTAL		
			CASES	PER CENT	
3 to 9	5	3	8	1.6	20.0%
10 to 19	6	7	13	2.6	
20 to 29	9	13	22	4.4	
30 to 39	25	32	57	11.4	
40 to 49	45	44	89	17.8	
50 to 59	75	45	120	24.0	
60 to 69	72	36	108	21.6	
70 to 79	44	22	66	13.2	
80 to 89	10	3	13	2.6	
90	2	2	4	0.8	
Total	293	207	500	100	

was of interest, therefore, to determine whether age and the degree of atherosclerosis of the aorta were well correlated. The age limitation set by most banks would infer a close relationship.

Degree of Aortic Sclerosis Related to Sex and Age.—The gross description of the entire aorta was reviewed in all cases. In many there was a comparable degree of atheromatous degeneration throughout the vessel. In some, however, this was more marked in the lower abdominal aorta. Such an observation is not surprising, since this is the site of most arteriosclerotic aneurysms, which would develop presumably in the area of maximal degeneration. In a rare case, sclerosis was greatest in the thoracic aortic arch. The microscopic findings in a representative section of aorta were also noted. The most severe degree of degeneration, whether by gross or microscopic examination, was then recorded for each individual case. The changes were listed as Grade 0 if no signs of degeneration were present, Grade 1 if there was intimal or sub-

TABLE IV. CORRELATION OF THE DEGREE OF AORTIC SCLEROSIS WITH SEX AND AGE FROM FIVE HUNDRED AUTOPSIES

(Presbyterian Hospital, May, 1949, to May, 1950)

	AGE (IN YEARS)										TOTAL		
GRADE	3 TO 9	10 TO 19	20 TO 29	30 TO 39	40 TO 49	50 TO 59	60 TO 69	70 TO 79	80 TO 89	90	CASES	PER CENT	
						293 Men							
0	5	5	1	4			*1*				16	5.5	16.5%
1		1	2	7	8	*7*	*7*				32	11.0	
2			*5*	8	27	34	21	*15*	*1*		111	37.5	
3			*1*	*5*	6	20	17	7	4	1	61	21.0	46.0%
4				*1*	*4*	14	26	22	5	1	73	25.0	
Total	5	6	9	25	45	75	72	44	10	2	293	100.0	
						207 Women							
0	3	5	4	2		*1*					15	7.3	25.6%
1			5	15	11	*5*	*1*			*1*	38	18.3	
2		*2*	*3*	15	26	22	16	*6*	*1*		91	44.0	
3			*1*		5	12	15	6	1	1	41	19.8	30.4%
4					*2*	5	4	10	1		22	10.6	
Total	3	7	13	32	44	45	36	22	3	2	207	100.0	

Grade 0—No evidence of degeneration
Grade 1—Intimal or subintimal thickening
Grade 2—Atheroma
Grade 3—Definite atheroma and calcification
Grade 4—Ulceration

intimal thickening, Grade 2 if actual atheroma was present, Grade 3 if calcification had developed, and Grade 4 if ulceration of the aorta was present (Table IV).

Only Grade 0 and Grade 1 were considered as being completely acceptable for graft material. It was thought, however, that many cases classified as Grade 2 with slight, patchy atheromas, would have certain segments of aorta which could be considered as Grade 1, and hence acceptable. It was interesting to note that there were only 48 vessels of Grade 0 and Grade 1 among the 293 men, or 16.5 per cent. Among the 207 women, there were 53 such instances or 25.6 per cent. Conversely, there were 134 vessels with advanced degeneration among the men, or 46.0 per cent, as compared to 63, or 30.4 per cent, among the women. This simply indicates the occurrence of a greater degree of atherosclerosis among men.

However, if one next correlates the degree of aortic degeneration with age, there are more than a few which deviate from the standard trend (in italics, Table IV). Among the men, there were a total of 31 patients over the age of 50 years in whom the degeneration was much less than would be expected. Fifteen of these had Grade 0 and Grade 1 vessels. One outstanding example was a 64-year-old man, dying shortly after an operation for a localized brain tumor. No significant degeneration was noted in his entire aorta. Among the women, there were 15 cases with minimal degeneration over the age of 50 years.

On the other hand, there were patients under the age of 50 years with unusual degeneration. Among the men, there were 16 such cases, and 12 were less than 40 years of age. As might be expected, premature atherosclerosis was less frequent among the women. There were only 8 patients under the age of 50 with excessive aortic degeneration, and 6 of these were women less than 30 years of age.

Degree of Aortic Sclerosis Related to Cause of Death.—With variations in anticipated sclerosis noted, it was of interest to see if there was any correlation of aortic degeneration with the causes of death. Such an analysis readily showed that certain conditions were associated with maximal or minimal atherosclerosis (Table V). Classification was sometimes difficult since a single cause was to be listed. In general, the major consideration was given the factor which might be of most importance in the selection of vessels for grafts. All patients with neoplasm were so listed, even if pulmonary embolism may have caused death, as occurred in 5 cases. Another patient dying of pulmonary embolism was listed primarily as subacute bacterial endocarditis (generalized infection) because this condition would preclude the use of the vessels. A death from mitral stenosis was similarly classified as subacute bacterial endocarditis. This portion of the analysis concerns only patients having autopsy within 24 hours.

A. Cardiovascular lesions (140 cases, 28 per cent): As might be expected, patients dying of cardiovascular lesions usually had a moderate to marked degree of aortic sclerosis, with only 43.0 per cent having minimal degeneration. The largest number of cases in this group had death caused by

myocardial infarction. Only 25 per cent of 44 cases had Grade 2 degeneration and there was only 1 case with Grade 1. The young individuals with excessive aortic sclerosis were mostly patients dying of occlusion of the coronary arteries. Other cases with moderate to marked degeneration were those dying of advanced generalized arteriosclerosis, rupture of aneurysm of the abdominal aorta (21 cases), and of advanced essential hypertension (32 cases). There were about one-half of the latter group with aortic degeneration of Grade 2 or less. Of 19 patients dying of cerebrovascular accident, one-half had excessive sclerosis of the aorta. In general, the remainder died of rupture of a congenital cerebral aneurysm. By far the best vessels among the cardiovascular cases were found in patients dying of peripheral or pulmonary embolism, congenital heart disease, and mitral stenosis. Two patients dying of mitral stenosis are not included in this category because of active rheumatic fever or subacute bacterial endocarditis.

B. Neoplasm (212 cases, 44 per cent): This group was subdivided because some men have suggested the use of vessels from patients with neoplasm localized to the central nervous system. We were surprised to note that in the group with brain tumors, 82 per cent of the 38 cases had little aortic degeneration. One-half of them had only Grade 0 and Grade 1 degree of aortic sclerosis. As was expected, the other 174 cases with neoplasm were usually in the older age group. Among these, 63 per cent had moderate to little degeneration, with only 31 cases (18 per cent) showing Grade 0 and Grade 1 changes. In general, it was observed that minimal sclerosis was present in patients dying with brain tumor or leukemia. In the latter, abnormal cells were often found in the wall of the vessels. Of interest were 5 cases with neoplastic cells noted in the adventitial vessels of the aortic section. The primary malignancies were sarcoma and carcinoma of the lung and breast.

TABLE V. DEGREE OF AORTIC SCLEROSIS; CORRELATION OF CAUSE OF DEATH WITH AORTIC DEGENERATION OF A MINOR DEGREE*

CAUSE OF DEATH	TOTAL	GRADE 0 TO GRADE 2	
		CASES	PER CENT
Cardiovascular	140	60	43
Neoplasm (excluding brain tumor)	174	111	63
Brain tumor	38	33	82
Degenerative	40	26	65
Endocrine	6	4	67
Infection, general	28	19	68
Renal	20	14	70
Shock and hemorrhage	12	9	75
Infection, local	11	9	82
Miscellaneous	13	11	85
Total	482	296	63 average

*Autopsy performed within 24 hours of death.

C. Degenerative conditions (40 cases, 9 per cent): In this category are patients dying of cirrhosis, emphysema, amyloidosis, and central nervous system degeneration. In only the latter were the vessels very good, although there were 26 cases with Grade 0 to Grade 2 degeneration in the entire group. The majority of cirrhotics had Grade 2 changes.

D. Endocrine conditions: This small group of 6 cases includes 4 with myasthenia gravis and 2 with Addison's disease. The vessels were moderately good in these patients.

E. Infection:

General (28 cases, 6 per cent).—In these cases are listed deaths from septicemia, poliomyelitis, subacute bacterial endocarditis, and syphilis. Certain cases of syphilis have been classified under other categories such as neoplasm which would in general preclude use of vessels. Almost 70 per cent of the patients dying with generalized infection had less than excessive degeneration of the aorta.

Local (11 cases, 2.5 per cent).—In this classification are patients dying of pneumonia, peritonitis, abscess of the brain, or other viscera. The vessels were not considered satisfactory if in the region of the infection, but it was thought that at least one segment might be obtained elsewhere. Since a strict bacteriologic check is made of all vessels in the bank, there would be little chance that contaminated material would be used. In this group there was little sclerosis present, only 18 per cent having excessive degeneration.

F. Renal conditions (20 cases, 4 per cent): There were 20 patients dying primarily of uremia resulting from nephritis, malignant nephrosclerosis, or polycystic kidneys. A case of periarteritis nodosa with uremia was not listed here, because the vascular disorder would be more important in the selection of grafts. The vessels were remarkably good in 7 and Grade 2 in another 7 cases.

G. Shock and hemorrhage (12 cases, 2.5 per cent): The cases of those dying of shock were mostly of postoperative patients (4 cases) and 1 burn case. In this group, the vessels were very good in all but 2. The patients dying of hemorrhage were not accident cases, but those suffering of bleeding from the esophagus or gastrointestinal system. A few were postoperative. Most of these cases had Grade 2 degeneration.

H. Miscellaneous (13 cases, 2.5 per cent): In this group were placed a scattering of cases which were probably unsuitable for grafts. They include blood dyscrasia, periarteritis nodosa, disseminated lupus erythematosis, and pemphigus vulgaris. Also included were cases of intestinal volvulus and silicosis. In general, the vessels showed little advanced degeneration.

Comment.—In considering the causes of death, which would render a donor unsuitable, the most frequent question concerns the use of an otherwise satisfactory vessel from a patient dying of a localized malignancy. Until all the factors which can produce malignancy are better understood, extreme caution should be exercised in the consideration of the use of such material. If one were to employ such donor material, it was evident in our analysis that patients dying of brain tumors would seem to be the most suitable candidates.

A similar problem is faced in the consideration of patients who had active rheumatic fever, periarteritis nodosa, lupus erythematosis, and other widespread mesenchymal diseases. The question of using tissues from individuals dying of advanced endocrine disturbances or blood dyscrasias, has not been answered. Most authorities would agree that aortic tissue should not be

obtained from patients dying of generalized infection such as subacute bacterial endocarditis, septicemia, miliary tuberculosis, syphilis, or even poliomyelitis.

It would seem, therefore, that extreme caution should be exercised in every single case, excluding for the present time any individual with a condition which may be transmissible or in which the etiologic agent is not thoroughly understood. Consequently, it would still seem wiser to explore the possibilities of expanding the limitations on age and time period after death, rather than the causes of death. The analysis of these autopsies did reveal certain facts which may be of importance in evaluating these possibilities.

As was expected, a greater degree of aortic sclerosis was noted in men, but the degree of degeneration did not always correlate with the age in either sex. In a significant number of cases of both sexes, there were patients over fifty years of age with vessels which would have been suitable on the basis of the minimal degenerative changes. Certain causes of death seemed to be associated with a greater degree of aortic sclerosis. Patients dying of myocardial infarction, for example, showed the greatest amount of degeneration, regardless of the age at the time of death. It has been thought by some that patients dying a sudden death should be an excellent source for vessel grafts. However, some statistics have shown that the most frequent cause of sudden death (65 per cent) is actually myocardial infarction,[23] with its accompanying high degree of aortic sclerosis.

It should be mentioned, moreover, that young individuals meeting sudden accidental death through trauma or violence have been thought to be perhaps the best donors for vessel grafts.[21] Such cases did not make up a part of our 500 autopsies, since emergency ambulance service is not a function of this hospital. There are certain important facts, nevertheless, which should not be overlooked in the determination of acceptability of such cases. It would seem important to emphasize the possibility of the presence in such cases of active syphilis, or of an unsuspected condition which should preclude the use of the vessels.

The proper handling of such individuals dying of violence is of great importance because of the many factors which influence post-mortem deterioration.[23, 24] Unless early refrigeration is carried out, certain undesirable changes begin to develop soon after death. The environmental temperature and amount of clothing influence this rate of deterioration. An individual with an obese body habitus requires greater speed and care in handling than a thin person. Because of the increased body size and water content of the tissues, the slower fall in body temperature permits more rapid and severe post-mortem changes. It is interesting to note the very slow rate of deterioration of stillborn infants. This is believed to be due to sterility of the gastrointestinal tract and to the rapid rate of cooling of the body. Thin individuals lose body heat rapidly and infants even more so, owing to the greater surface of the body in proportion to its size. An average adult body at a normal room temperature loses about 2° F. of body heat per hour during the first few hours after death, and thereafter about 1° F. per hour.[23] Old individuals,

dying of a wasting disease, may actually lose body heat before death. Such a patient, whose tissues are dying before the individual dies, has been found to have less desirable aortic tissue.[21] At ten hours a significant amount of internal body heat remains, even though the surface has cooled. It would appear to be very important, therefore, that bodies be refrigerated as soon as possible after death if vessel material is to be obtained from them. Certainly, in sudden deaths by violence, especially where detection of the victim of a criminal assault may be delayed, the period after death may be appreciable or even unknown before refrigeration of the body. Even in fatal street accidents, the time required for police investigation, examination by a medical examiner, and the subsequent journey to the morgue may be much more than that required in a hospital before refrigeration.

Interested in determing the rate of deterioration of aortic tissue at varying periods after death, we have conducted experiments since early October, 1950, which might yield such information, since we could not derive it from an analysis of autopsies.

Methods.—In the first set of experiments, two series of adult mongrel dogs were studied. A large dose of pentobarbital sodium was injected intravenously to cause death within a few seconds. After the hair was shaved from the chest and abdomen, the skin was cleaned and aseptic preparation with tincture of iodine and 70 per cent alcohol was performed. The field was draped with sterile towels and a vertical skin incision was made from the upper end of the sternum to just above the pubis. Large sterile sheets were sutured to the wound edges before the chest or abdomen was entered. Upon entering these cavities, the left lung and abdominal viscera were reflected to the right. The diaphragmatic leaf on the left was divided, and full exposure of the aorta was thus achieved. The vessel was carefully mobilized in its bed so that subsequent manipulation of the vessel would be minimal.

At this time, a segment of aorta measuring 1.5 to 2 cm. in length was resected for control studies, most often from the thoracic aorta below the subclavian artery. A bacteriologic culture was made of the aortic bed at this time. The viscera were then replaced into normal position and a sterile towel, moistened with 0.8 per cent saline solution, was placed in the open wound to cover the viscera. The sterile sheets were then folded over the sterile field so that they would cover the animal and yet the incision could be re-exposed without contamination. After appropriate intervals, identical segments of aorta were obtained and bacterial cultures taken. The body was refrigerated during the entire period of study unless noted otherwise.

Group I consisted of 6 animals which were refrigerated in toto at 4° C. immediately after the control aortic segment and culture were taken. Thirty-five subsequent specimens, not including the controls, were obtained from 2 hours post mortem to a maximum of 168 hours (7 days), or an average of 6 segments per dog. In 2 animals these specimens were obtained during a 30 hour period. In 2, the total interval was 72 hours and 96 hours, respectively. In the final 2, the period was 168 hours.

Group II consisted of 6 animals which were left at a room temperature of 25 to 26° C. before refrigeration. In 1, the interval was one hour, and, in the other 5 dogs, the period before refrigeration was 4 to 6 hours. Twenty-four specimens were obtained after securing the control segments. The dog kept at room temperature only 1 hour was studied for a total of 240 hours (10 days). Of the others, 3 were studied for 48 hours, and 2 for 168 hours (7 days).

The rate of loss of body temperature was studied in this group, by careful and frequent check of the rectal temperature following death.

In all but the first animal, tissue culture was carried out by the method described previously.[14] A 4 mm. ring of aorta was sectioned so that separate colonies, 1 mm. on a side, could be imbedded on the bottom of a Carrel flask in a clot of 33 per cent chicken plasma. Usually two flasks were prepared, each containing 16 colonies. After the plasma clotted, a supernatant fluid consisting of 33 per cent dog plasma and 5 to 10 per cent dog spleen extract in Simms' X6 solution was added. Five per cent carbon dioxide gas in air was added to the flask, which was then incubated at 37° C. The average amount of cellular growth observed in the two flasks was taken as the growth rating for the specimen. Observations of the multiple colonies were made daily for a period of several weeks, and alterations in the growth behavior and morphology were noted. These details will be published separately, since only the presence of cellular growth is of importance in this report.

The remainder of each aortic segment was then processed for orthodox histologic study as well as for special histochemical preparations. The former was accomplished by placing the tissue in Bouin's fixative for at least 12 hours and then transferring it to 80 per cent ethyl alcohol. The cut sections were stained with hematoxylin and eosin for general structure and cellular detail, with Verhoeff's stain for elastic fibers, and Masson's trichrome stain for smooth muscle cells and connective tissue. In some instances Laidlaw's stain for reticulum fibers was used.

The preparation of aortic tissue for histochemical studies was more complex. Since the quick-freezing or lyophilizing techniques of fixation were not yet available to us, chemical fixation was carried out.

A 2 mm. aortic ring is passed through 2 changes of saline-formalin solution before imbedding in Carbowax. This tissue was then suitable for study of the lipid fractions. Sudan black B revealed fats or complex lipids as a black staining substance.

A second aortic ring was placed into alcohol-formalin solution, and refrigerated at 4° C. After passage through absolute alcohol at both 4° C. and room temperature, the segment was kept under cedarwood oil. This tissue could be used to show the structural components by orthodox stains, if desired. Toluidine blue 0 stain was used on similar sections to demonstrate the distribution and character of the mucopolysaccharides by virtue of their metachromasia. They are revealed as a purple staining substance, possibly functioning as the lubricant of organs rich in connective tissue. They are mainly hyaluronic acid, chondroitin sulfate, and heparin. Certain sections

were incubated an hour with hyaluronidase before staining. The hyaluronic acid was thus removed, and only chondroitin and heparin were demonstrated. In other sections incubated 12 hours before staining, the chondroitin was also removed, leaving only the heparin. These residues stained purple as did the original complex substance, but were progressively less in quantity. When tissue fixed in alcohol-formalin was processed with periodic acid, carbohydrates formed periodioaldehydes. The Schiff reagent produces a red color when combined with aldehydes. Glycogen and mucopolysaccharides can thus be shown by the characteristic vermilion color. If similar sections are incubated with diastase before processing, the glycogen is removed.

A third ring of aorta was refrigerated in acetone, and after 2 changes was kept under cedarwood oil at 4° C. until it was processed. The tissue was treated by the Gömöri technique for the demonstration of alkaline phosphatase. Phosphate is liberated and combines with calcium ions supplied in a substrate. Cobalt ions are added to replace the calcium and cobalt sulfide is formed in the areas orignally containing alkaline phosphatase. This black substance shows well with the red safranine counterstain.

RESULTS

A. Loss of Body Heat.—The normal rectal temperature of a series of adult dogs of small and medium size (4 to 12 kilograms) was 38 to 39° C. When the body remained in an environmental temperature of 25° C., with the top surface covered by a folded sheet, there was a fall in rectal temperature of 2° C. in as short a time as 45 minutes. At 1 hour, the loss was usually from 3 to 3.5° C. During the second hour, there was moderate variation, a further fall of from 1 to 3° C. being recorded. Subsequently, however, the rate slowed to 1 to 1.5° C. fall per hour up to 6 hours. With refrigeration at 4° C., the body temperature was equilibrated to 4° C. by 24 hours.

B. Bacteriologic Studies.—In Group I, cultures were made throughout four experiments, being completed at 30, 72, 96, and 144 hours, respectively. In none was there a positive culture. This was further confirmed by the absence of bacterial contamination in any of the tissue cultures. In the cultures of one animal, a yeast was found, and this was traced to a single vial of dried chicken plasma.

In Group II, cultures were obtained during the study of 4 animals, the last ones being at 48, 48, 120, and 168 hours, respectively. All of these dogs were kept at room temperature for over 4 hours before refrigeration. Positive cultures were obtained in the 2 long-term studies. In one, *Staphylococcus aureus* was found at 48 hours, and a Clostridium at 72 hours. The cultures obtained at 120 and 168 hours were negative. In the other animal, the 24 hour culture revealed *Bacillus coli* and an alpha streptococcus. Subsequent cultures revealed alpha streptococcus or *B. coli* and *Cl. welchii.* The control cultures obtained in these 2 dogs were negative. Tissue cultures of the aortic segment from these dogs were satisfactory and did not reveal gross bacterial contamination. This was of interest because, in our previous experience, unsterile aortic tissue has been very unsuitable for tissue culture owing to early bacterial overgrowth.

C. Tissue Culture Studies.—Aortic tissue was studied throughout 5 experiments of Group I. Because of the growth pattern, the results of the first animal in Group II are included in those of Group I. This dog was refrigerated one hour after death, and there were no differences noted from the group cooled immediately after death.

The results in 2 of the other experiments were unsatisfactory. In one, the control tissue showed excellent growth, but the contamination of the chicken plasma by a yeast made observations of the other cultures impossible. In the second dog, control tissue grew well, but the failure of subsequent tissues to grow was attributed to the use of instruments soaked in Zephiran. This agent is very toxic to tissue cells, even in very high dilution. Hence, satisfactory results were obtained in 4 of 6 animals.

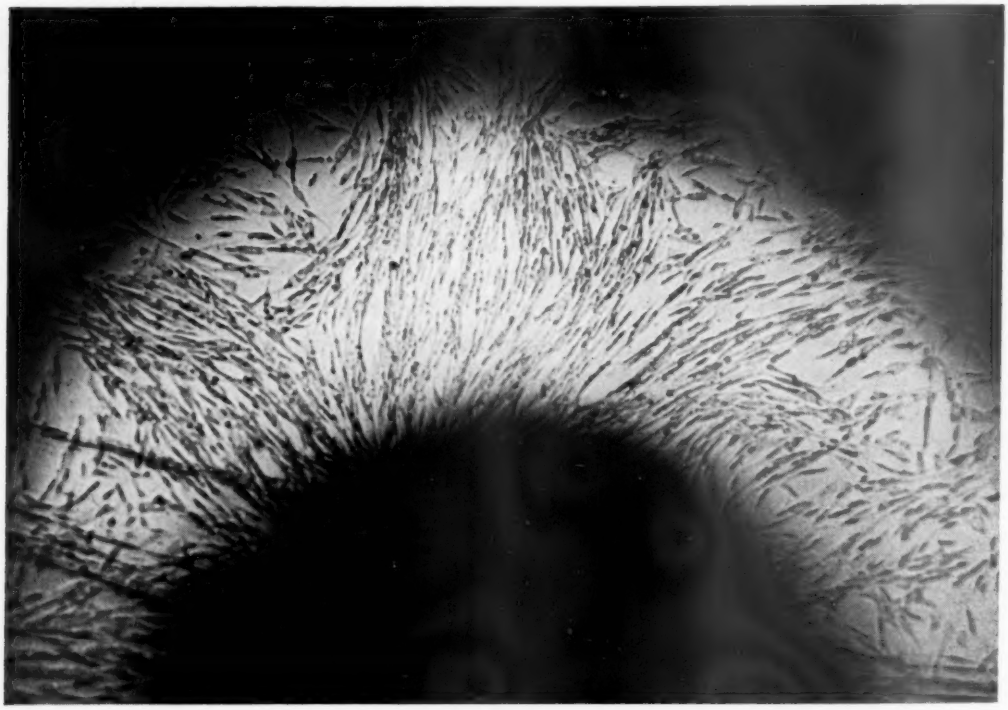


Fig. 1.—A 15 day living tissue culture of aortic tissue removed from a dog refrigerated after death for 7 days (×150, unstained).

Fibroblastic growth was excellent in all of the control tissue, obtained at time of death. Definite growth was noted throughout the entire experiment in the 4 dogs, the final specimen being obtained at 48 hours post mortem in 2, at 168 hours in 1 (Fig. 1), and 240 hours in the last. There was a definite diminution in the amount of growth after 48 hours, with very scanty growth and with delay in appearance of cells in the 240 hour specimen (Fig. 2, *A*).

In Group II, 4 dogs refrigerated at 4 to 6 hours after death were studied. The final specimens were obtained at 48 hours in 2 and at 168 hours in 2. Control tissue grew well in all instances. There was a definite decline in the

amount of cellular growth at 24 hours, scanty growth at 120 hours, and none at 168 hours (Fig. 2, *B*).

D. Orthodox Histology.—

Group I: In those animals refrigerated immediately after death, there was a very slow rate of degeneration. There was no significant alteration from normal control structure until after 24 hours. (Fig. 3, *A* and *B*). Then slight

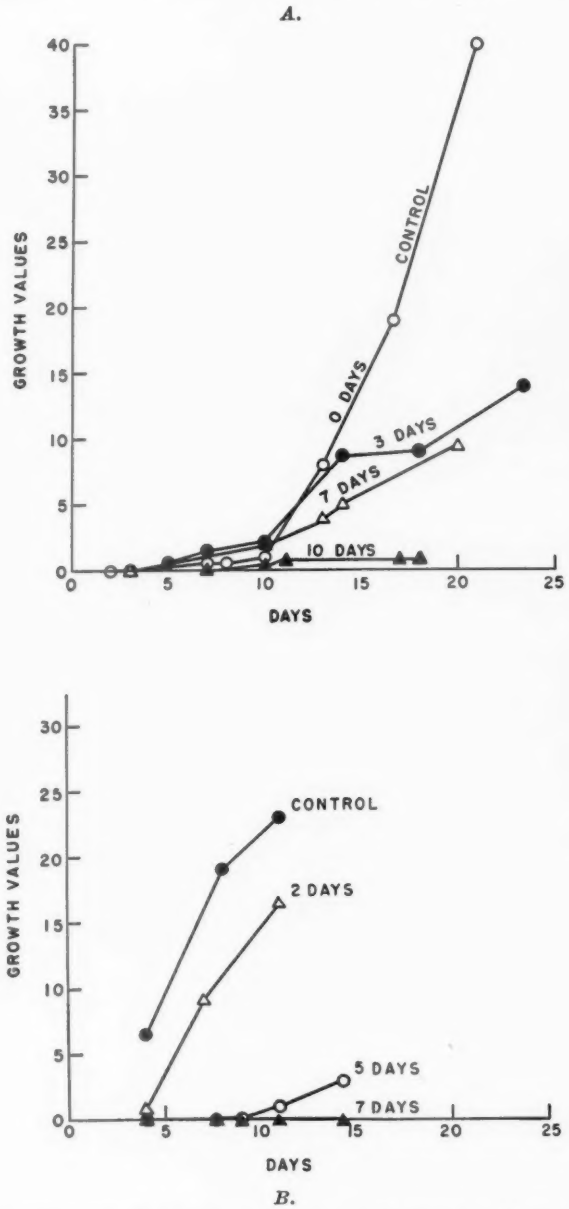
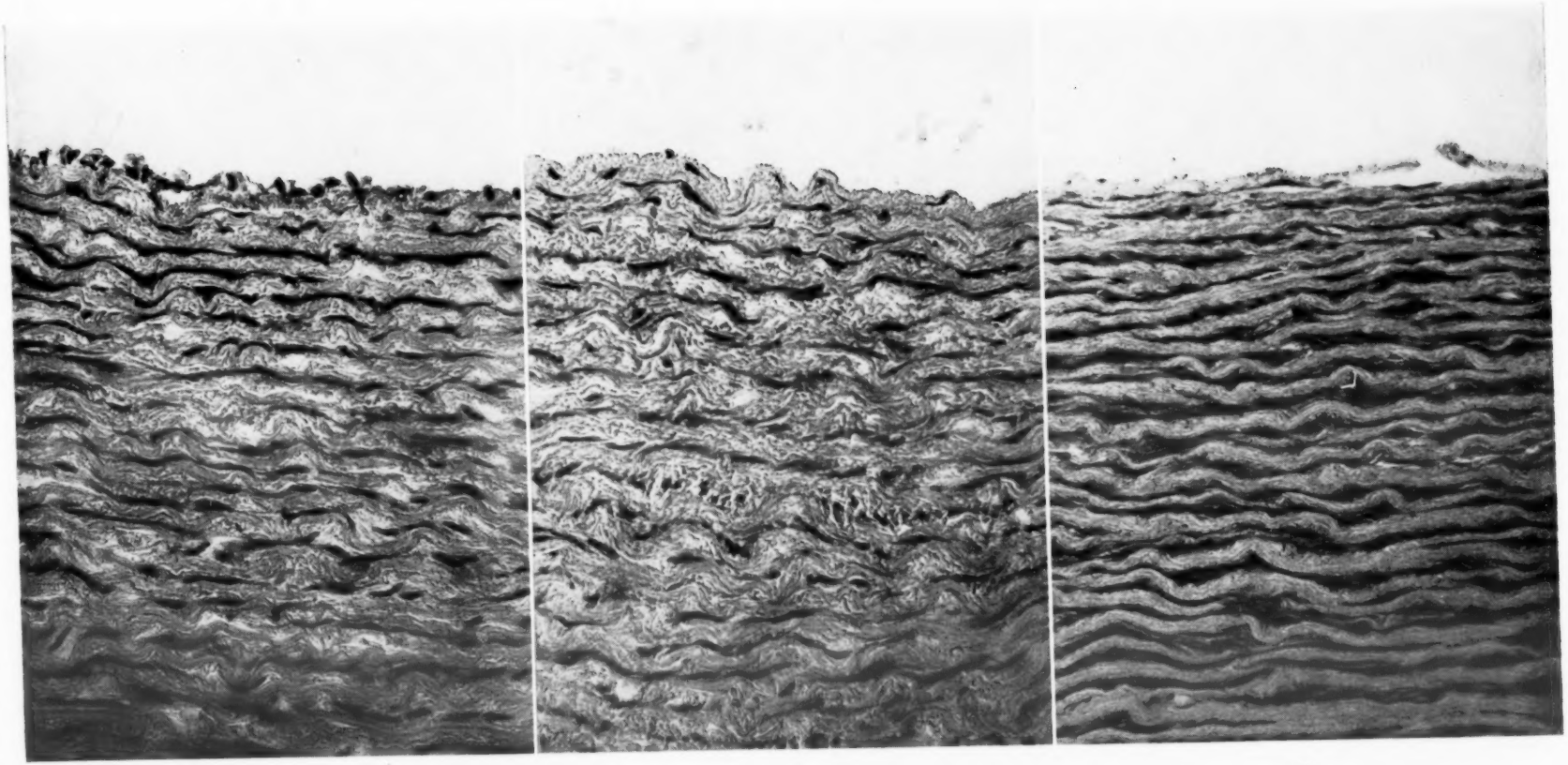


Fig. 2.—Graphic representation of typical results of tissue culture of dog aorta.

A. Growth values of cultures of aortic tissue obtained at the time of death, and after refrigeration of the body at 4° C. for 3, 7, and 10 days.

B. Growth values of cultures of aortic tissue obtained at time of death and after refrigeration at 4° C. for 2, 5, and 7 days. An interval of 4 hours at 26° C. preceded the period of refrigeration.



A. *B.* *C.*

Fig. 3.—Histologic appearance of aortic tissue from dog (trichrome stain, ×225), *A*, at time of death (control). *B*, After 48 hours of refrigeration of the body at 4° C., minimal degenerative changes are noted. *C*, After 120 hours of refrigeration there is marked pyknosis of the nuclei, disappearance of smooth muscle and endothelium, and a condensation of the elastic framework.

darkening of the nuclei was occasionally noted, although not until 48 hours in some animals. By 48 hours, definite degenerative signs were found in the smooth muscle cells of the media, and at 72 hours, the endothelium was ragged. There was definite pyknosis of the nuclei. Some muscle cells were gone from the media, and most of the others showed definite degeneration. By 96 hours, there was less endothelium. Disappearance of muscle cells was moderate and pyknosis of the nuclei was marked. At 144 hours, there was clumping of nuclear material in many areas. The endothelium was gone and the media was vacuolated. The wall of the vessel showed some thinning, being partly the result of condensation of the cellular debris between the elastic fibers (Fig. 3, *C*). By 168 hours these changes were still more advanced. Some areas of the media no longer contained smooth muscle. In none of these sections were the elastic fibers significantly altered.

Group II: The changes described above occurred more rapidly as the period of time before refrigeration lengthened. In the animal refrigerated 1 hour after death, there was no difference from the findings in the dogs of Group I. However, if over 4 hours elapsed after death before refrigeration, there was a definite acceleration of signs of degeneration. At 6 hours post mortem, there was slight pyknosis noted. By 24 hours, this had increased and degeneration of endothelium and smooth muscle had begun. At 48 hours, the endothelium was patchy, the smooth muscle thinned, and moderate pyknosis was noted. Vacuolization and thinning of the wall began at 72 hours, with marked changes noted beyond this time. By 168 hours, there appeared to be a patchy loss of elastic fibers in the media with marked thinning of the vessel wall.

E. Histochemical Changes.—

1. Alkaline phosphatase was present in the control sections only in the inner intimal zone, and in the nerves and small vessels in the adventitia. None was observed in the media.

In Group I, this material persisted only for a few hours, being gone in most instances by 6 hours. In Group II, this substance had disappeared an hour or two after death.

2. Mucopolysaccharides, including heparin, hyaluronic acid and chrondroitin sulfate, were present in large amounts in the control sections. In discrete strandlike collections, this material was distributed between the collagen bundles of the media, being mostly in the inner media. A lesser amount was noted in the intima, beneath the internal elastic membrane. Very little of this material was found in the adventitia. After the incubation studies to demonstrate heparin, the tissue revealed most heavy concentrations beneath the elastic membrane of the intima.

In Group I animals, there was very slight diffusion of the material noted about 24 hours after death, possibly due to depolymerization. This diffusion was definite at 48 hours, with perhaps some disappearance of the substance beginning at 72 hours. There was a definite diminution in metachromasia observed at 96 hours, with marked diffusion of the residual substance. The scanty material remaining at 144 hours had disappeared by 168 hours.

In Group II, the process was accelerated. Some diffusion had begun at even 6 hours with more advanced alteration and slight disappearance noted at 24 hours. Very little of this material remained at 120 hours.

3. Glycogen was present as discrete vermilion granules in the intima and media of control sections. In Group I, there was a tendency to clumping of the granules by 24 hours, and some coalescence and disappearance by 48 hours. By 72 hours, there was little if any glycogen. None was present at 96 hours or longer. In Group II, the coalescence was definite by 6 hours, and there was moderate loss by 24 hours. None was noted after 48 hours.

4. Studies of lipids were too few to draw conclusions as to pattern of degeneration and disappearance. In the few animals studied in both groups, there was no change in the lipids throughout the entire experiments.

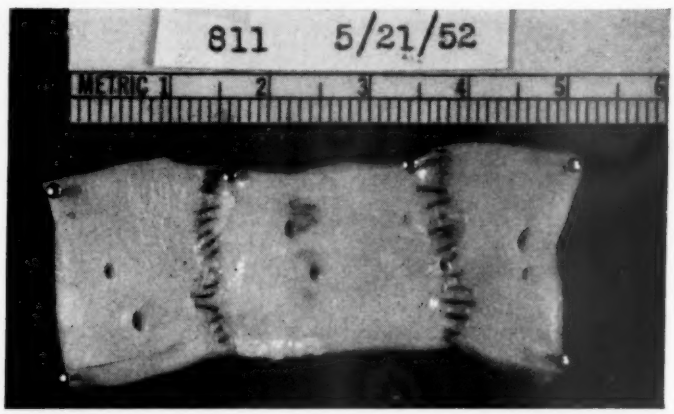


Fig. 4.—Aortic homograft obtained from a dog which had been kept 4 hours at 26° C. after death and then refrigerated at 4° C. until 48 hours post mortem. This graft was stored in a nutrient electrolyte solution at 4° C. for 26 days and had been implanted in another dog for 35 days.

COMMENT

These experimental studies revealed definite information regarding the rate of degeneration of aortic tissue after death. If the animal was refrigerated almost immediately after death, bacterial contamination was not observed. The histologic structure remained almost normal for 24 hours, with the more advanced degeneration occurring after 48 hours. Tissue culture revealed fibroblastic growth through 10 days after death, but there was a reduction in the amount of growth after 48 hours. The histochemical studies correlated well. Alkaline phosphatase remained only a few hours, glycogen began to deteriorate at 24 hours, and the mucopolysaccharides were also altered by 24 hours.

If there was a delay in refrigeration of 4 to 6 hours after death, these changes occurred much sooner, as might be expected. In order to test the functional state of vessels obtained under aseptic conditions a long time after death, a final set of experiments was performed.

Method.—A medium-sized adult dog was killed by intravenous pentobarbital sodium. After being in an environmental temperature of 26° C. for 4

hours, the intact body was refrigerated at 4° C. At 48 hours post mortem, the aorta was exposed under aseptic conditions and 4 aortic segments measuring 3 cm. each were obtained for grafts. These were stored in Simms' X6 solution containing 10 per cent homologous serum for from 16 to 26 days. Two of the grafts were then implanted into the low thoracic aorta and two into the abdominal aorta of other dogs. A continuous suture of 00000 braided arterial silk was used for the anastomoses. A segment of aorta was resected from the host animal before implantation of the grafts. No anticoagulant drugs were employed. These animals have been observed for over 3 months.

Results.—In all the dogs, function remained excellent. Two animals were killed at 1 week and 2 months (Fig. 4). There was no thrombus nor gross signs of degeneration. The microscopic pattern did not vary from that seen in grafts handled in accordance with present vessel bank criteria[15] (Fig. 5, *A* and *B*). Aortograms revealed normal function in the 2 surviving animals, but one was completely calcified at 10 months. The other graft appeared excellent, grossly, at 10 months.

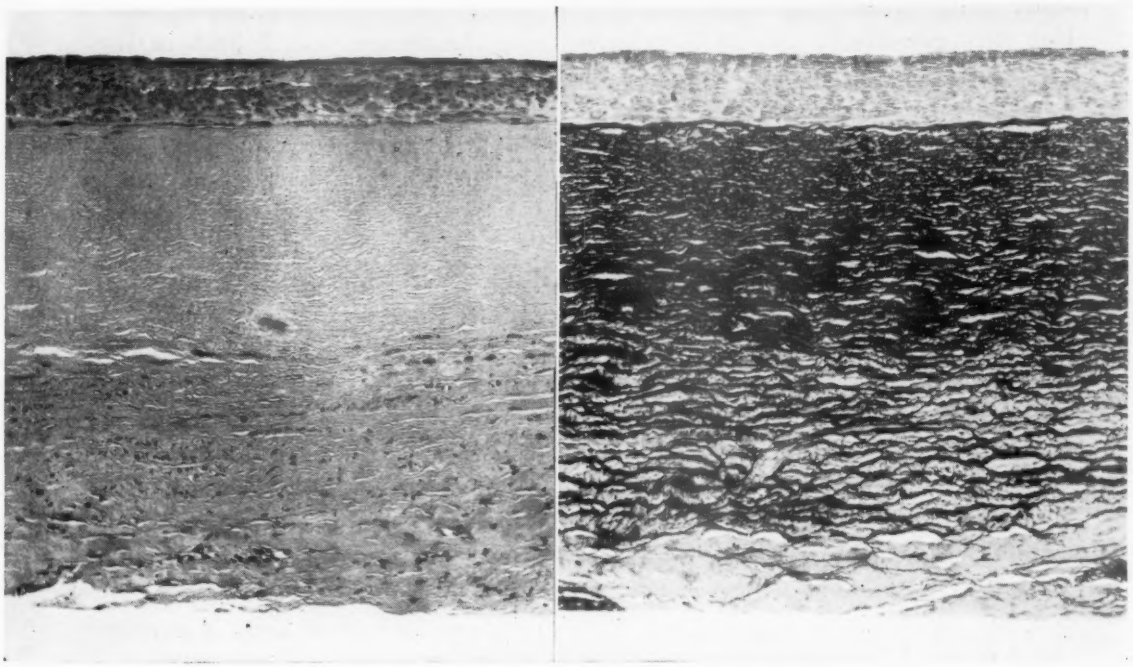


Fig. 5.—Photomicrographs of the aortic homograft shown in Fig. 4.

A, There is new fibrocellular intima from the host animal covering an acellular layer which represents the media of the implanted graft. New fibroblasts have invested the adventitial layer. (Hematoxylin and eosin, ×40.)

B, The elastic fibers of the homograft are condensed, but intact. (Verhoeff, ×40.)

DISCUSSION

From the information obtained from this analysis of autopsies and from the experiments on dogs, it would seem that certain of the present strict criteria governing the selection and use of preserved arterial homografts may indeed be modified to allow access to a larger group of donors. It would ap-

pear that the physical age of an individual is a better index of the anticipated degree of aortic sclerosis than the chronological age. The body habitus plays some part in influencing the rate of cooling after death, and hence in the relative rate of deterioration of the tissues. The cause of death may also suggest the probable condition of the aorta, degeneration being greatest in the individuals dying of myocardial infarction. Keeping these points in mind, one might reasonably select certain cases in the older age group, with a good chance of obtaining satisfactory vessels.

In all likelihood, the most inflexible restrictions should still be imposed in the consideration of acceptable causes of death or medical conditions. Experience has shown that vessels obtained from individuals dying of penetrating wounds or sustaining ruptured viscera are usually contaminated.[21] The presence of significant amounts of blood in the thoracic or abdominal cavities similarly enhance the chance of contamination. An interesting correlation was noted between certain findings of the New York Blood Vessel Bank and our experimental studies. Positive cultures have been obtained occasionally from the pleural or peritoneal cavity of human donors at the time of autopsy but with repeatedly negative cultures from the vessels. In 2 dogs of Group II, positive cultures were similarly obtained from the thoracic and abdominal cavities. In both, satisfactory tissue cultures were obtained from the aorta with no evidence of bacterial contamination. In our past experience the tissue culture matrix has proved to be an excellent medium for bacterial growth. Since antibiotic drugs were not employed in the tissue culture preparation nor in the temporary storage solution, it is not clear why there has been this discrepancy in bacterial activity. Possibly, the initial concentration of organisms was so low that the dilution factor alone explains the absence of demonstrable contamination of the vessels. It is of practical interest that no positive cultures were obtained from the animals refrigerated immediately after death. This fact emphasizes the desirability of early refrigeration after death. The tissue-culture, histological, and histochemical studies fully support this. While electronic irradiation of grafts insures sterility, the finding of late calcification in vessels implanted after use of high doses of irradiation would make adherence to strict aseptic technique advisable for the present time.[17] Experiments in this regard are being continued.

We should give more attention to the length of time a body is unrefrigerated than to the lapse of time after death before vessels are procured. The latter source of concern arose from the results of early experiments in this field. Gross and his co-workers[8] performed studies on grafts which had been kept moist at 4° C. If the period of storage was less than 6 hours following death, these grafts were successful. Thrombosis or rupture occurred in most instances if the grafts were kept for from 18 to 78 hours before they were used. Recommendations were made that the period after death during which the most satisfactory vessels could be obtained should not exceed 6 hours.[10] The authors stated "beyond six hours, we felt there was a likelihood of rapid appearance of post mortem change and of bacterial contamination."

In another group of dogs, excellent results were obtained by the same authors with grafts stored at 4° C. from 2 to 98 days in a balanced electrolyte solution. Attention was called to the demonstration of viable cells in these grafts during the first 6 weeks of storage.[8] In a subsequent publication[9] viability was considered to be an important factor in assuring functional success, in view of the discouraging results with nonviable frozen grafts. On the basis of those studies, limitations were suggested which we feel may now be relaxed. Greater consideration should be given to the factors of body habitus and rate of cooling of the body, as well as of the physiologic age, disease entities, and cause of death of the donor.

Properly handled, a well-selected body should supply very satisfactory vessels for a reasonably long period after death. Seven grafts obtained from human donors between 16 and 35 hours after death have been used in New York with completely satisfactory results.* The details of these cases will be published in the near future.[25]

SUMMARY

From many experiments on dogs since 1949 and from an analysis of 500 autopsies performed at Presbyterian Hospital in New York, certain facts have been derived concerning the procurement, storage, and use of aortic homografts.

1. Satisfactory results have been obtained with nonviable grafts preserved by quick freezing or lyophilization or stored for over 40 days in a nutrient electrolyte solution.

2. Viability of cells in a graft is not an essential factor in assuring functional success.

3. The acceptable age of a donor individual should be estimated from his physiologic appearance and cause of death rather than from stated age.

4. Individuals with a cause of death or associated condition which may be transmissible or about which the etiology is not fully understood should be excluded as donors at the present time.

5. Emphasis should be directed toward rapid refrigeration of the body with less concern being exhibited over the interval of time between death and procurement of vessel grafts. Aseptic technique should still be employed when obtaining these vessels.

6. Suitable donors, properly handled, should yield satisfactory grafts for a period well beyond 6 hours after death. Grafts obtained from 16 to 35 hours after death have been used clinically from the New York Vessel Bank with success.

The silk arterial suture material was generously donated by J. A. Deknatel and Son, Inc. We wish to acknowledge the valuable suggestions of Dr. George H. Humphreys, II, and the active participation of Drs. Claude C. Coleman, Jr., Kenneth K. L. Hui, Edward von Hasseln, and of Marvin Mufson and Peter Westerhoff in this study. The cooperation of Dr. Henry P. Smith of the Department of Pathology is greatly appreciated. The opinions expressed in this paper are not necessarily those of the New York Vessel Bank Committee, but have played a part in the formulation of the present criteria governing the selection and use of preserved blood vessel grafts in this bank.

*From the New York Vessel Bank.[12]

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A NONSUTURE AORTIC SHUNT—AN EXPERIMENTAL STUDY

ROBERT J. IZANT, M.D., CHARLES A. HUBAY, M.D.,
AND WILLIAM D. HOLDEN, M.D., CLEVELAND, OHIO

(From the Departments of Surgery, Western Reserve University School of Medicine and University Hospitals)

BECAUSE of recent advances in surgical techniques applicable to surgery of the vascular tree and the current improvements in diagnostic methods, a greater number of vascular lesions, which previously were deemed inoperable, are presenting themselves for therapy. Both clinically and experimentally the procedures used for attacking vascular problems have progressed rapidly to the point where the problems of surgical intervention per se are no longer as hazardous as previously considered. Accompanying the need for unhurried and meticulous surgery is the equally important need, on occasion, for establishing an adequate diversion of blood flow about a completely occluded area. Our aim was to devise a method for the purpose of permitting sufficient flow around an occluded area in the thoracic aorta.

It seemed to us that the prerequisites of a satisfactory arotic shunt should include: (1) simplicity of design, (2) ease of introduction into and removal from the aorta, (3) sufficient lumen to permit adequate blood flow, and (4) enough security to prevent leakage and accidental dislodgment. If fulfilled, these criteria would permit the use of a shunt to prevent the irreversible damage which often occurs from cross-clamping the thoracic aorta for fifteen minutes or longer.

After considerable experimentation with various materials, a shunting instrument was constructed utilizing double-lumen, double-flanged, stainless steel tubes with an inside diameter of 5 mm. (Fig. 1). The inner flange is inserted into the thoracic aorta through a small linear incision and the outer flange is approximated by means of a threaded knob, thus holding the aorta securely enough between the two flanges to produce a "water-tight" seal. A second identical instrument is inserted in a similar fashion at a lower level in the aorta (Figs. 2 and 3). The two shunts are then connected by means of plastic tubing.

It has been pointed out by numerous investigators[1-7] that complete occlusion of the thoracic aorta for longer than twenty-five to thirty minutes is accompanied by a high incidence of hind-quarter paralysis and/or irreversible shock. Because of this it was considered worth while to perform two series of experiments on dogs to compare survival with the aorta occluded to survival with the aorta occluded but with our shunt functioning.

EXPERIMENTAL PROCEDURE

Two series of five dogs each were operated upon. The dogs were all healthy adult mongrel dogs weighing between 15.4 and 22.4 kilograms. Intravenous

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sodium pentobarbital in a dose of 30 mg. per kg. was used as anesthesia. An endotracheal tube was introduced and open-chest respiration was maintained by a positive pressure mechanical respirator. Under aseptic conditions, a left thoracotomy was performed through the bed of the seventh rib. Intercostal arteries from T_4 to T_7 were ligated and the adventia removed from a convenient length of midthoracic aorta. In the first group of dogs, the midthoracic aorta was cross-clamped for forty-five minutes.

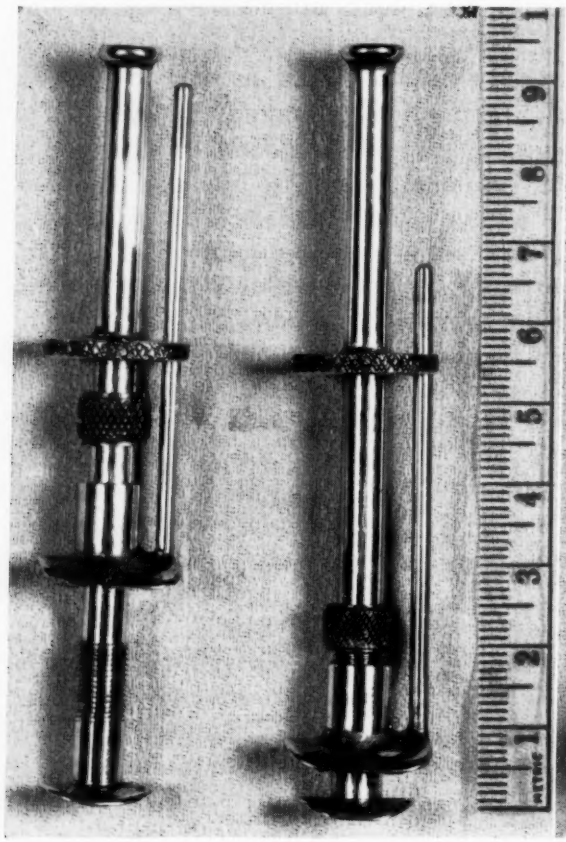


Fig. 1.

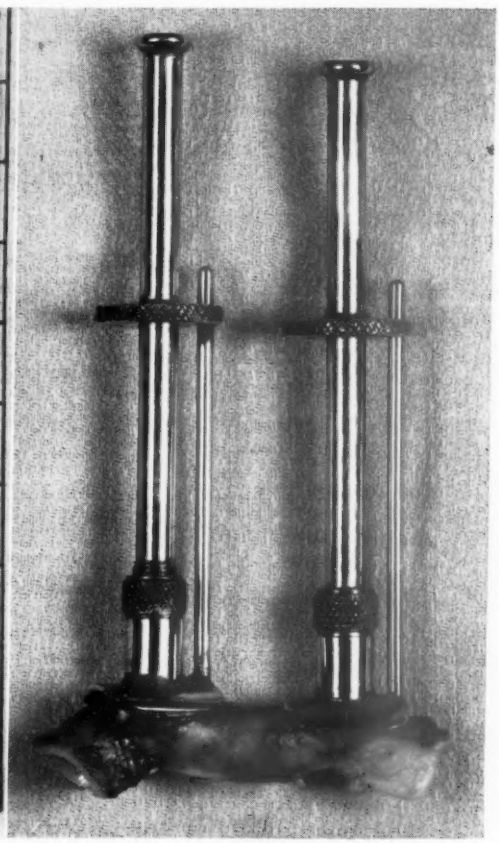


Fig. 2.

Fig. 1.—Aortic shunting instruments. Right, flanges closed. Left, flanges apart.
Fig. 2.—Shunting instruments in position in the midthoracic aorta of a dog.

A second group of five dogs were similarly operated upon with the exception that the occluded area was by-passed by inserting the shunting device above and below the point of occlusion. One to two minutes of complete occlusion were necessary to insert and to remove each shunt. The two shunts, previously treated with Arquad 2 C,* a nonwetting agent, were connected by means of a polyethylene tube with a 5 mm. bore. The aorta between the shunt was cross-clamped with two Potts coaretation forceps and divided transversely. The aorta was then anastomosed end to end by means of a continuous everting suture of 00000 arterial silk. The aorta was occluded forty-five to sixty minutes.

*Kindly supplied by Armour and Company, Chicago, Ill.

The longitudinal incisions in the aorta were then closed by means of a continuous 00000 arterial silk suture utilizing Beck's partial aortic occlusion clamp[8] which maintained a dry field and simultaneously allowed moderate aortic blood flow. The chest was then closed in routine fashion. No antibiotics or anticoagulants were used in either series.

In other healthy adult mongrel dogs shunts were inserted in a similar manner in the thoracic aorta. Cannulas were also placed in the carotid and femoral arteries to record, manometrically, pressures on a kymographic record. In some animals, bladder urine formation was measured by inserting a mushroom catheter through a suprapubic wound into the urinary bladder. In three animals the left renal artery pressure was measured.

In two experiments the rate of flow through the unobstructed midthoracic aorta was compared to the rate of flow through the shunt when the aorta was occluded. These determinations were obtained by means of a large capacity rotameter.[9*]

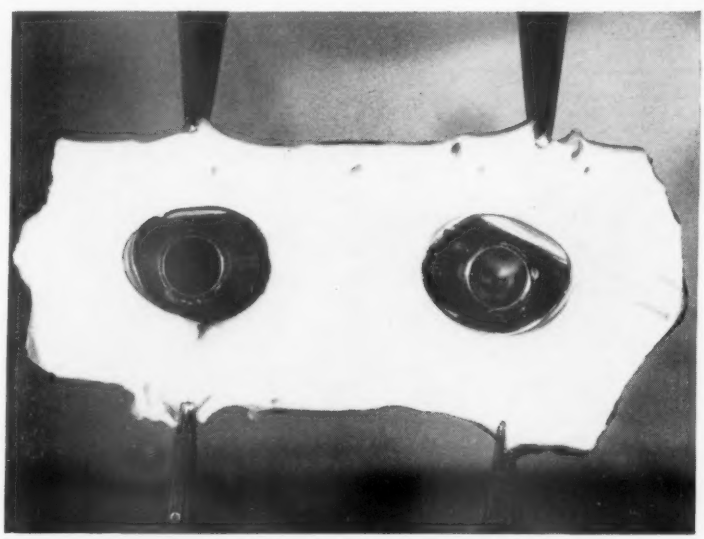


Fig. 3.—View from inside aorta of shunting instruments in place in the midthoracic aorta of a dog.

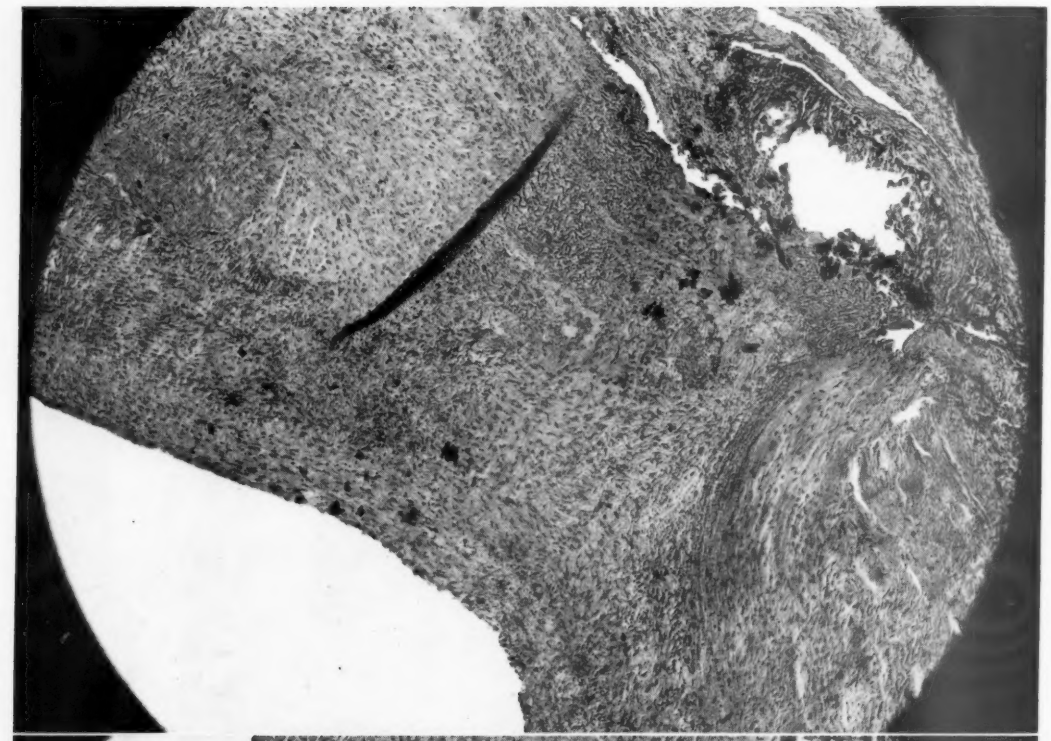
RESULTS

In the control group of five dogs whose midthoracic aortas were cross-clamped for forty-five minutes, two died and two had permanent hind-limb paralysis. Two of these dogs that died within twenty-four hours of operation had autopsy evidence of submucosal intestinal hemorrhage similar to that seen in dogs dying with irreversible hemorrhagic shock.

None of the five dogs at autopsy showed complete occlusion of the aorta because of thrombosis at the site of cross-clamping. However, all showed minor amounts of intimal damage. One dog with hind-limb paralysis (AS-16), sacrificed at the end of seven days, had a small mural thrombus (2 mm. in

*We are indebted to Dr. Richard W. Eckstein of the Department of Medicine, who performed these studies.

Fig. 4.



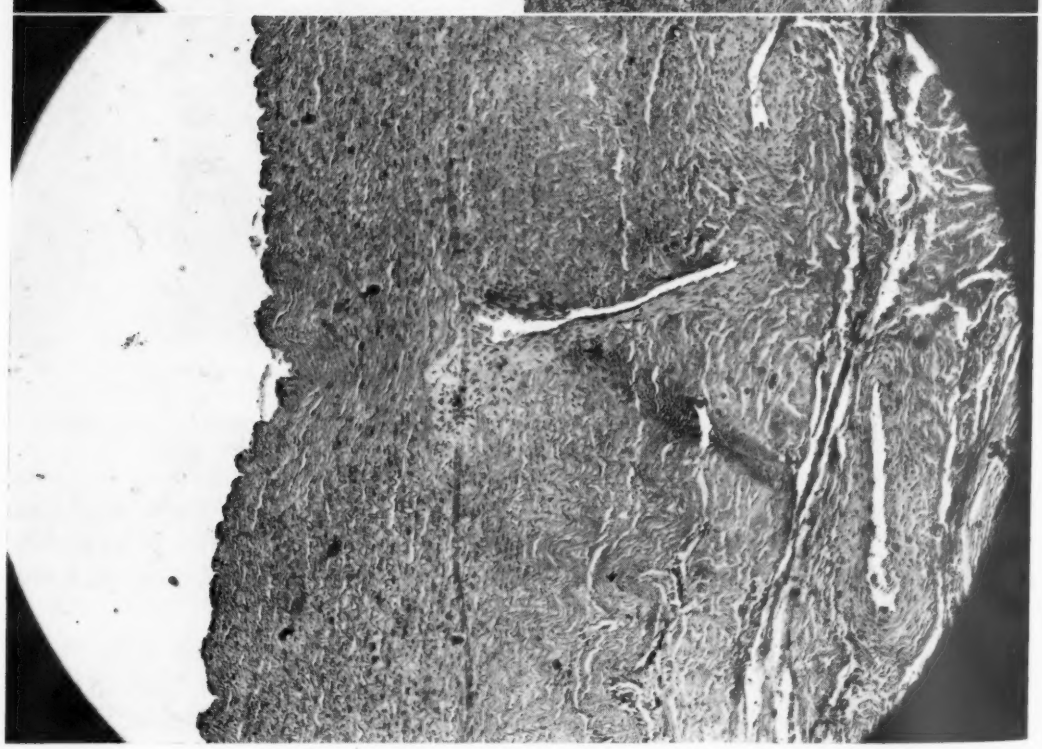


Fig. 5.

Fig. 4.—Photomicrograph at point of anastomosis of midthoracic aorta showing intact intima.

Fig. 5.—Photomicrograph of midthoracic aorta at site of insertion of shunting instrument.

diameter) arising at the point of initial damage and trailing freely down the aorta for a distance of 5 cm. This thrombus occluded only a very small portion of the aortic lumen.

In the experimental group of five dogs, the midthoracic aorta was cross-clamped for forty-five to sixty minutes. During this time collateral circulation was maintained by the shunt while anastomosis of the aorta was carried out. None of these dogs developed hind-limb paralysis, nor did any dog die from shock. One dog died five days postoperatively from empyema and pneumonia. All dogs in this group were autopsied from six days to two months after the experimental procedure. Microscopic study of the aorta after two months showed complete restoration of the intimal linings not only at the site of aortic anastomosis, but also at the site of the insertion of the shunting instruments (Figs. 4 and 5). In all instances there was a moderate amount of fibroblastic proliferation about the operative site, but examination of the areas which had been in contact with the flanges of the shunt failed to show any pathologic alteration. No evidence of postoperative leak or aneurysmal dilatation was found.

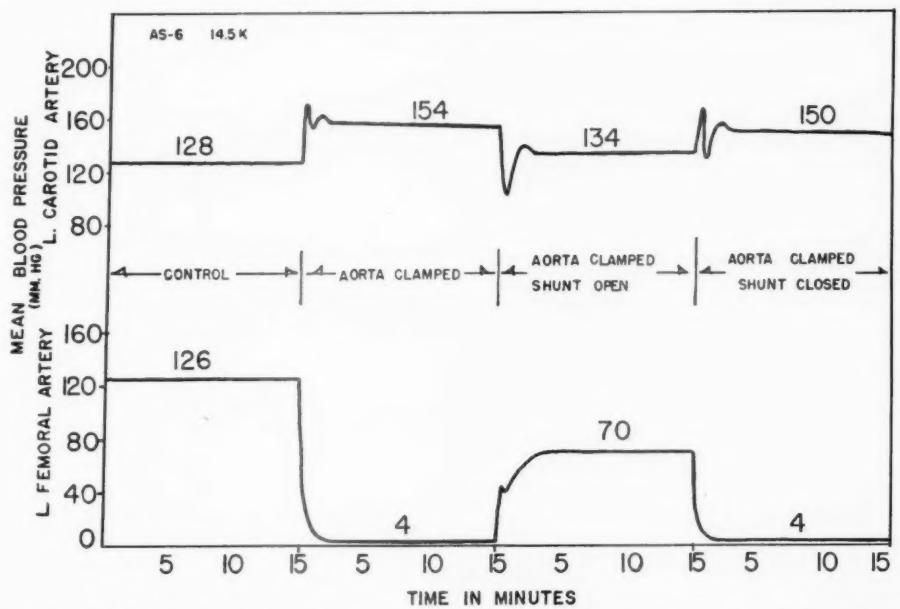


Fig. 6.—Effect of shunt on the mean carotid (upper line) and femoral (lower line) artery blood pressures.

In animal AS-6 (Fig. 6), the aortic shunt was inserted and the carotid and femoral pressures were recorded. After the shunt was in place, but not functioning, the control mean carotid and femoral pressures were 128 and 126 mm. Hg, respectively. The aorta was then cross-clamped with the shunt closed and the mean carotid pressure stabilized at 154 mm. Hg. The mean femoral pressure fell abruptly to 4 mm. Hg. After fifteen minutes the shunt was opened and the femoral arterial pressure rose and stabilized at 70 mm. Hg. The carotid pressure fell to 134 mm. Hg. Under similar circumstances in another animal,

AS-21, the mean femoral pressure ranged between 81 and 94 mm. Hg when the shunt was functioning and the midthoracic aorta was cross-clamped.

In animal AS-29 (Fig. 7), the aortic shunt was inserted and the left carotid and left renal artery pressures were recorded. The control carotid and renal pressures were 154 and 150 mm. Hg, respectively, with the shunt in place and the aorta open. The aorta was then occluded between the shunt and the carotid pressure stabilized at 174 mm. Hg, while the left renal artery pressure fell to 80 mm. and stabilized at 82 mm. Hg. The shunt was then also occluded and the renal artery pressure fell to 18 mm. Hg, the carotid pressure rising to 200 mm. Hg. After three minutes the shunt was reopened and the renal artery pressure rose to 82 mm. Hg. Upon reopening the aorta the renal and carotid pressures returned to control levels.

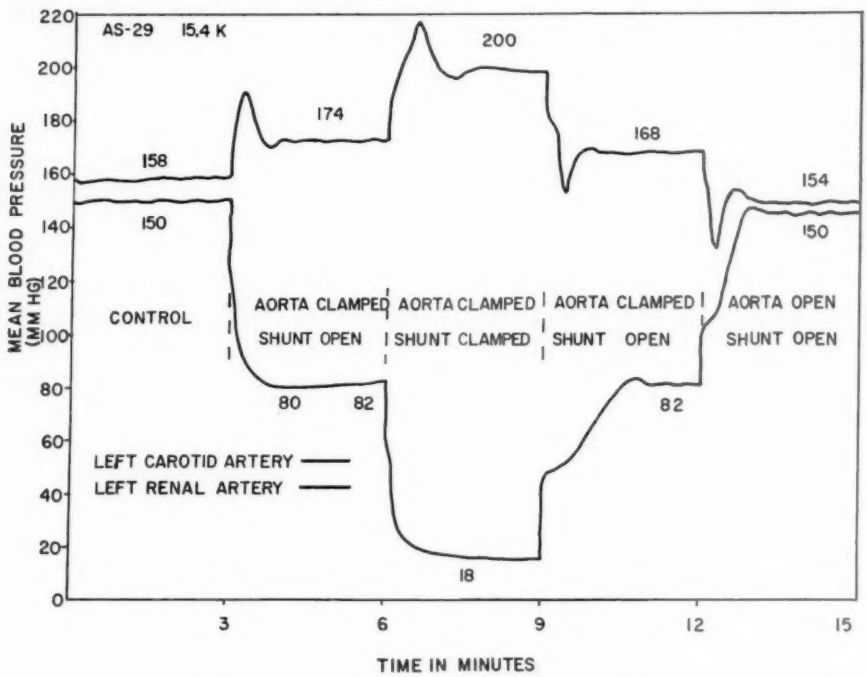


Fig. 7.—Effect of shunt on mean carotid and renal artery blood pressures.

In our studies on the rate of urine formation in animal AS-21 (Fig. 8), 1.8 c.c. of urine per minute was collected under control conditions. With the shunt functioning and the midthoracic aorta occluded, urine formed at the rate of 0.77 c.c. per minute. In the recovery phase, 0.82 c.c. per minute was obtained. This but slight increase may be due to the fact that not enough time was allowed for complete recovery of the kidney to take place. After a fifteen- to twenty-minute period of reduction of the effective renal filtration pressure, it may be thirty to fifty minutes before complete recovery occurs.[10] However, with both the aorta and the shunt occluded, urine formation practically ceased (0.07 c.c. per minute).

The rotameter studies showed that with a mean *aortic* pressure of 113 to 125 mm. Hg, the blood flow through an unobstructed dog aorta was between

910 c.c. and 1,045 c.c. per minute. With the aorta occluded and the shunt functioning, the flow through the shunt varied between 770 c.c. and 843 c.c. per minute. With the shunt in place, the mean femoral pressures obtained simultaneously with the shunt-flow study were 76 to 93 mm. Hg. The mean *aortic* pressure during the period that the shunt was functioning did not deviate appreciably from the control levels.

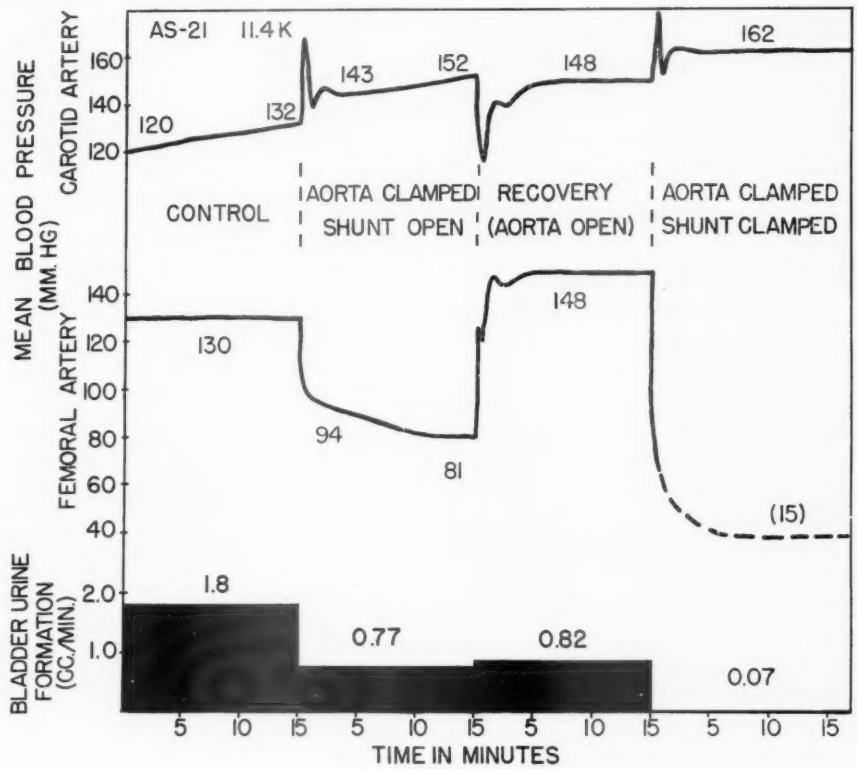


Fig. 8.—Effect of shunt on mean carotid and femoral artery pressure and bladder urine formation.

DISCUSSION

As early as 1910, Carrel[11-13] pointed out the problems involved in occluding a previously unobstructed thoracic aorta for more than ten to fifteen minutes. Later Blalock,[1] Gross,[2] DeCamp,[7] Clatworthy,[5] and Schafer[6] re-emphasized the problem and demonstrated that dogs with aortas so occluded develop permanent hind-limb paralysis or die of shock in a high percentage of cases. The observations in our small series are similar (Table I).

In cases of adult-type coarctation with sufficient previously established collaterals, prolonged occlusion of the thoracic aorta does not present this problem to so great a degree as do lesions without previously established collaterals, i.e., aneurysms, thrombosis, or neoplasms involving the aorta. However, a coarctation of the aorta in a 19-year-old white man, who was operated upon at the University Hospitals of Cleveland, was brought to our attention recently.[14] In the patient, the left subclavian artery arose just distal to the coarcted area

TABLE I. SUMMARY FROM THE LITERATURE (INCLUDING PRESENT SERIES) OF SURVIVAL STUDIES AFTER CROSS-CLAMPING THE MIDTHORACIC AORTA, WITH AND WITHOUT SHUNTING DEVICES

AUTHOR	TIME AORTA OCCLUDED	NORMAL SURVIVAL	DIED IN 24 HOURS OR HAD HIND-LIMB PARALYSIS
Gross and Hufnagel*			
16 dogs	30 to 60 minutes	4	12
12 dogs	4½ to 30 minutes	8	4
Schafer and Hardin			
13 control dogs	25 to 50 minutes Average, 32 minutes	1	12
11 experimental dogs	45 to 180 minutes Average, 107 minutes	11	0
Clatworthy and Varco			
13 control dogs	Not recorded†	3	10
Izant, Hubay, and Holden			
5 control dogs	45 minutes	1	4
5 experimental dogs	45 to 60 minutes	5‡	0

*Excluding dogs sacrificed at end of procedure or whose hind quarters were packed in ice.

†Clatworthy and Varco did not record the time the aorta was occluded except to indicate that it was greater than thirty minutes.

‡1 died of pneumonia on the fifth postoperative day. Not paralyzed.

so that the artery had to be ligated in order that the coarctation could be resected. The aorta was occluded for forty-five minutes. It was noted that (1) blood flow was from the subclavian artery into the aorta and (2) the intercostal arteries were not so hypertrophied as would be expected. The operation was well tolerated; but permanent, though mild, lower leg weakness has required the use of braces. The time necessary to produce permanent ischemic changes in the lumbar spinal cord in a human being is not known and may be only remotely related to the times observed in experimental animals.

Various attempts at by-passing occluded areas in major vessels including the aorta have been made. Carrel first used a permanent intraluminal paraffin-coated tube.[13] Hufnagel[15] used a plastic tube. Smith[16] suggested a soluble rod as an aid in anastomosis, but did not report experiments on the aorta. DeCamp[7] modified the use of the intraluminal tube in that it was used only as a temporary expedient, but this still required longer periods of complete occlusion than is safe. Poth's splint[17] requires temporary occlusion also. Clatworthy and Varco[5] used an experimental shunt which required the sacrifice of the subclavian artery; not a desirable feature for clinical application. The experimental approach of Blalock[1] required the sacrifice of the left subclavian artery. Schafer and Hardin[6] developed small-bore (1.14 to 2.69 mm.) polyethylene shunts for both experimental and clinical application. But these are less secure than desirable, and in some instances more than one shunt was necessary. In abstract form, Leeds and Culnier[18] report the use of peripheral arteries above and below the occluded area, maintaining circulation by means of a roller-type pump. It is assumed that sacrifices of arteries would be required in the procedure.

There appears to be litle doubt, however, as to the necessity for a shunting device about a totally occluded area of the midthoracic aorta provided no natural collateral system pre-exists. The question of survival of an animal under these experimental conditions mentioned seems to hinge on whether the pressure distal to the occluded area in the midthoracic aorta is maintained above a

critical level, probably 60 to 70 mm. Hg. This prevents ischemic changes in the spinal cord and kidney as reported by Gross[2] and Selkurt.[10] The latter has pointed out that creatinine clearances of 50 per cent or better are obtained when the kidney is perfused at a pressure of 60 to 70 mm. Hg. Below this pressure clearances rapidly drop to low levels. With our shunting apparatus in place and functioning and the aorta occluded, we recorded pressures from 70 to 94 mm. Hg in the femoral artery; in another experiment, renal artery pressure ranged between 80 and 82 mm. Hg (Fig. 7).

One of the best indices for adequate renal blood flow is urine formation. The glomerular filtration of urine ceases when the systemic arterial pressure is progressively lowered. This critical pressure is in the neighborhood of 70 mm. Hg.[19] Corcoran and Page[20] and Selkurt[10] have shown that irreversible changes in renal function occur in dogs if the period of hypotension exceeds forty minutes, during which time appreciable renal damage is produced.

Under the conditions of our study, with the shunt functioning and the femoral artery pressure between 81 and 94 mm. Hg, there was adequate urine formation. We feel justified in concluding that, since urine was being formed, there was sufficient collateral circulation through the shunt to maintain an effective glomerular filtration pressure. On the basis of the observations it would appear that the shunting device described allowed sufficient perfusion of blood at adequate pressures to supply at least minimal physiologic renal requirements.

Shipley and Gregg[21] have pointed out that reduction of the lumen of a blood vessel may not cause physiologic alterations in the vascular bed it supplies if the rate of flow through that vessel is low. However, should the rate of flow through that vessel become greater because the demands of that vascular bed increase, the same amount of constriction may produce serious metabolic changes. Also, the degree of reduction of flow due to external constriction of the lumen of a vessel cannot be predicted. Accordingly, a comparison of the flow through the shunt with the flow through the open aorta was necessary. This showed that with the size shunt being used there was only a 15 to 20 per cent reduction of blood flow. Simultaneous mean femoral pressures were well above the critical level of 60 to 70 mm. Hg. Also, it is interesting to point out that no significant alteration in mean *aortic* pressure occurred while the shunt was functioning.

Gupta and Wiggers[22] demonstrated that a decrease of 60 per cent in the lumen of the thoracic aorta was necessary before significant alterations of the ventricular pulse pressure curves or of the femoral and carotid pressures occurred.

It immediately becomes apparent that the nearer the lumen of a shunt approximates the lumen of the vessel being by-passed, greater flow and fewer physiologic alterations will occur. Work is now in progress to study the effects of a 1 cm. bore shunt.

CONCLUSION

From the evidence presented we feel that the new shunting device as described improves existing techniques and has certain advantages over present methods.

The manner of application together with survival studies in dogs has been reported.

Pressure and aortic flow studies, together with the rate of urine formation, reflect the physiologic efficacy of the shunt.

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THE EMERGENCY TREATMENT OF MASSIVE BLEEDING FROM ESOPHAGEAL VARICES BY TRANSESOPHAGEAL SUTURE OF THESE VESSELS AT THE TIME OF ACUTE HEMORRHAGE

ROBERT R. LINTON, M.D., AND RICHARD WARREN, M.D., BOSTON, MASS.

(From the Surgical Department of the Massachusetts General Hospital)

THE construction of a veno-venous anastomosis between the portal and systemic venous systems, either by an end-to-side splenorenal shunt with splenectomy, or a direct portal vein-to-vena cava anastomosis, has proved to be in the hands of those experienced in this type of surgery the most effective means of preventing hemorrhage from esophageal varices, both in patients with cirrhosis of the liver, and in those with so-called Banti's syndrome. The results obtained in fifty-one patients operated upon in our clinic[7] and followed from one to seven years, demonstrate clearly the effectiveness of these procedures, since in the entire group there has been only one patient who has succumbed to postshunt esophageal hemorrhage (Table I). Many patients, however, are still dying from exsanguinating esophageal hemorrhages before a shunt can be performed, especially in the group with portal cirrhosis, because it has been impossible to control the esophageal varices. A recent analysis of ninety-three patients admitted to the Massachusetts General Hospital in the five-year period from 1946 to 1950, because of bleeding esophageal varices, has revealed that sixty-five, or 70 per cent, have had intrahepatic portal bed block, secondary to portal cirrhosis, and twenty-eight, or 30 per cent, had an extrahepatic block or the so-called Banti's syndrome (Table II). Despite severe esophageal hemorrhages in many of the latter group, there were no deaths and it was possible in twenty-three, or 82 per cent, of them to perform some type of a portacaval shunt. These low mortality and high operability rates undoubtedly are the result of dealing with relatively young patients with essentially normal livers. The results in the cirrhotic group, on the other hand, leave much to be desired, since it was only possible to perform shunts in thirty-three, or 51 per cent, of them because thirty-two, or 49 per cent, died either directly or indirectly from hemorrhage from the esophageal varices before they could be prepared for shunt surgery. Exsanguination was the chief cause of death in twenty-three, or 72 per cent, of these cases. Massive esophageal bleeding in four, or thirteen per cent, of the others was a major factor in causing liver failure from which these patients died. The other five, or 15 per cent, had mild or slight hemorrhages, which were a contributing factor in their deaths, also, from liver failure.

These observations demonstrate clearly that an early, effective, and safe means of controlling the ruptured esophageal varix at the time of acute hemor-

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TABLE I. RESULTS IN FIFTY-ONE PATIENTS WITH BLEEDING ESOPHAGEAL VARICES FOLLOWED FOR ONE TO SEVEN YEARS AFTER SHUNT SURGERY, MASSACHUSETTS GENERAL HOSPITAL (1945 TO 1951 INCLUSIVE)

	NUMBER OF PATIENTS	NUMBER OF DEATHS	CAUSES OF DEATH		
			LIVER FAILURE	CEREBRAL HEMORRHAGE	ESOPHAGEAL BLEEDING
Extrahepatic	19	1	0	1 (4 years)	0
Intrahepatic	32	4	2 (8 and 19 months)	1 (19 months)	1 (1 year)
Total	51	5	2	2	1

TABLE II. CAUSES OF DEATH IN THIRTY-TWO PATIENTS WITH BLEEDING ESOPHAGEAL VARICES WITHOUT PORTACAVAL SHUNT SURGERY, MASSACHUSETTS GENERAL HOSPITAL (1946 TO 1950 INCLUSIVE)

CAUSES OF DEATH	EXTRAHEPATIC	INTRAHEPATIC
Hemorrhage alone	0	23 (72%)
Hemorrhage with secondary liver failure	0	4 (13%)
Liver failure with slight hemorrhage	0	5 (15%)
Total	0	32

rhage is necessary, so that many of the patients that are dying from uncontrollable bleeding may have the benefit of shunt surgery. The purpose of this paper is to review the emergency measures that are now in use and to describe a method that has been used in our hospital for the past two years. A number of emergency methods of controlling the bleeding esophageal varices have been recommended in literature, but in most instances they have been ineffectual or carry too high an operative risk to warrant their use. They include:

1. *Ligation of the Splenic Artery.*—This was first advocated by Blain[3] and more recently by others,[6] but in our hands it has been of little or no value in the patient with massive bleeding, although it has seemed to control the slow ooze from the varices in an occasional patient (see Table III and Case 1).

TABLE III. RESULTS OF SPLENIC ARTERY INTERRUPTION FOR BLEEDING ESOPHAGEAL VARICES* MASSACHUSETTS GENERAL HOSPITAL (APRIL, 1950†)

NUMBER OF PATIENTS	NUMBER WITH POSTLIGATION BLEEDING	NUMBER OF POSTLIGATION DEATHS FROM HEMORRHAGE	NUMBER FOLLOWED BY SHUNT	NUMBER WITH POSTSHUNT BLEEDING	NUMBER OF POSTSHUNT DEATHS
8	6 (75%)	2 (25%)	5 (62.5%)	1 (20%)	0

*The left gastric artery was interrupted in three of the patients.
†None has been performed since this date because of these poor results.

2. *Ligation of the Hepatic and Splenic Arteries.*—This procedure has been advocated by Rienhoff[10] and Berman,[2] but it is a relatively new procedure the value of which for emergency, or even definitive treatment of bleeding esophageal varices, has not been established as yet.

3. *Injection of the Esophageal Varices With a Sclerosing Solution.*—It was first advocated by Crafoord and Frenckner,[4] and later by Moersch.[8] This procedure in our clinic and in many others has proved of little value in the acute bleeding phase, so as a result it has been abandoned by the majority.

4. *Esophagogastrectomy and Total Gastrectomy.*—The former was recommended by Phemister and Humphreys,[9] and the latter by the same authors and also Wangensteen.[13] These procedures, unfortunately, are such major surgical operations that the majority of patients in their depleted condition cannot withstand either, especially as an emergency method of treatment. Baronofsky[1] has reported a mortality of 50 per cent in eight patients subjected to total gastrectomy which, it is believed, is a prohibitive mortality rate.

5. *Esophageal and Gastroesophageal Tamponade.*—Gastroesophageal tamponade was first reported by Westphal,[14] who used an intragastric balloon that was inflated after it was placed in the stomach to produce pressure on the vessels feeding the bleeding esophageal varices. Rowntree and associates[11] in 1947 reported the use of an intraesophageal balloon. Sengstaken and Blakemore[12] more recently have reported good results using a two-balloon tube, with one in the stomach and the other in the esophagus.

The single intragastric balloon has been used in our clinic in the majority of the patients and in a few instances, more recently, the double balloon tube has been tried. The former has been found very effective for the emergency temporary control of the bleeding. It also has the advantage that it can be made from the double lumen Miller-Abbott tube, using a 10 cm. length of thin-walled Penrose rubber drain as the balloon. After the tube with the balloon on it is passed into the stomach, it is inflated to about the size of a large orange, with about 500 to 600 c.c. of air being used. Then it is drawn up against the cardioesophageal junction and maintained in this position by traction with a two-pound weight attached to the end of the tube, which is preferably brought out through the mouth as recommended by White.[15] It is believed that the intraesophageal balloon is not necessary provided sufficient tamponade is produced at the cardioesophageal junction to obstruct the submucous veins in the stomach which connect and feed the esophageal varices. The amount of pressure applied to collapse these veins does not need to be excessive since the pressure within them at the most is only in the neighborhood of 40 mm. Hg. Even though the actual rupture of the varix may be 4 to 5 cm. above the cardioesophageal junction, bleeding can be controlled by this type of balloon using a two-pound weight for traction, as already noted. Our experience with this type of balloon tube has been satisfactory in obtaining immediate control of the bleeding varices, but after removal of the tube severe hemorrhage has occurred again too frequently before the patient could be prepared for shunt surgery (see Case 1). It is without question a lifesaving procedure, when used correctly, since exsanguination may occur so rapidly in these cases, even with multiple blood transfusions.

If the bleeding is controlled, the seal at the site of rupture is not very strong since it is dependent only on an intravascular thrombus. Too frequently it loosens or dissolves and hemorrhage starts again. Repeated hemorrhages are a serious threat to a patient's life, not only because of the loss of blood, but also because anoxia and hemorrhagic shock develop to produce additional liver damage in an already failing liver, so that not infrequently, though the bleeding may be controlled, the patient eventually dies from liver failure. For this

reason it is recommended that an emergency operation should be performed, after the bleeding has been controlled and the patient's blood volume has been restored, to suture the esophageal varices. This procedure results in a more lasting control of the varices, although not permanent, which permits a longer period without bleeding for preparation of the patient for shunt surgery. Blakemore[12] has recommended leaving the two-balloon type of tube in the stomach and esophagus for a period of a week or more, feeding the patient through a third lumen in the tube. It is our opinion that many of these patients are too sick to consider for shunt surgery at the end of such a short period of preparation. For that reason we believe a shorter period of balloon tamponade is to be recommended, but at the same time controlling the bleeding esophageal varices by the suture technique, so that patients may be fed normally, even permitting them to be discharged home to regain their strength before subjecting them to the more major surgical procedure of a shunt operation.

The following case report demonstrates how ineffective the usual methods of treating a severe case of bleeding esophageal varices secondary to cirrhosis of the liver may be, and that some other method of controlling these bleeding vessels must be developed in order to save the patient's life and also permit time to prepare him so that a portacaval shunt can be safely performed.

CASE 1 (L. B. No. 695843).—A white man, aged 47 years, was admitted to the Phillips House of the Massachusetts General Hospital on March 24, 1950, complaining of vomiting of blood intermittently for three weeks. He was a chronic alcoholic of thirty years' duration. Twenty-one months before his admission he was treated for a questionable bleeding duodenal ulcer at another hospital which in retrospect probably was bleeding esophageal varices, as he was told at that time he had a sick liver. Six months ago he had a transient episode of tarry stools. Three months ago he had severe hematemesis and tarry stools. Three weeks ago he was admitted to another hospital because of repeated hematemeses. He was treated there with balloon tamponade using the two-balloon type of tube. This was carried out for approximately a week with cessation of the bleeding. The tube was removed, but eight days before his admission to our hospital bleeding again occurred. While in that hospital he received twenty-four blood transfusions.

Physical Examination.—Physical examination showed a pale, sallow-complexioned well-developed, and nourished man who appeared very ill. The general physical examination was essentially negative except for spider angiomata in the skin and an enlarged liver and spleen. The blood pressure was 110 systolic and 60 diastolic.

Laboratory Examination.—The urine was negative for sugar and albumin. Hemoglobin was 7.5 Gm., white blood count 14,500, van den Bergh test 2.4 mg. per cent, prothrombin time 65 per cent of normal, total protein 4.69 Gm., albumin 2.88 Gm., globulin 1.81 Gm., and nonprotein nitrogen 45 mg. per cent. Because of continued bleeding the splenic and left gastric arteries were ligated and divided under ether anesthesia three days after his admission on March 27, 1950. Following this operation the patient stopped bleeding for a few days and his hemoglobin rose to 13.6 Gm. with daily transfusions, but on April 5, 1950, nine days after the operation, his stools became grossly bloody again and the following day his hemoglobin had dropped to 11.6 Gm. There had been a definite improvement in his serum protein by this time with a total protein of 6.73 Gm., albumin 3.47 Gm., and globulin 3.26 Gm., and the nonprotein nitrogen was 29 mg. per cent. On April 10, 1950, his hemoglobin dropped to 9.5 Gm. indicating further bleeding. On April 17, 1950, despite continued daily transfusions, the hemoglobin continued to drop and was 8.8 Gm. At this time he vomited considerable bright blood so cardioesophageal tamponade with an intragastric balloon was instituted. On April 18, 1950, the bleeding was not controlled completely so the balloon was

removed but reinserted two days later with cessation of bleeding. At this time the hemoglobin was 9.4 Gm., total protein 5.17 Gm., albumin 2.77 Gm., and globulin 2.40 Gm. On April 21, 1950, the patient died of liver failure.

Post-mortem Examination.—The post-mortem examination revealed portal cirrhosis of the liver and esophageal varices with massive gastrointestinal hemorrhage. The source of the bleeding was found to be a small opening about 2 mm. in diameter in one of the esophageal varices about 5 cm. above the cardioesophageal junction (Fig. 1).

Fig. 1 (Case 1).—A photograph of an autopsy specimen showing the opened lower end of esophagus and upper end of stomach. The tip of the probe lies in the 2 mm. opening of the ruptured varix through which the patient exsanguinated. This large varix was a continuation of a huge submucous vein in the cardia of the stomach. This case first suggested the possibility of suturing the varices at the time of acute hemorrhage.

Comment.—This man, aged 47 years, demonstrates the difficulty in controlling esophageal bleeding in some patients with cirrhosis of the liver by means of the generally accepted measures. In this case double balloon tamponade had been temporarily successful before his admission to our hospital, but when he was admitted his condition was too critical to warrant a shunt procedure especially since his serum albumin level was only 2.88 Gm. and the nonprotein nitrogen elevated to 45 mg.[7] Interruption of the splenic and left gastric arteries was performed in an attempt to control the bleeding but it had little if any effect. Finally, despite daily transfusions, it was impossible to keep his hemoglobin above 8 or 9 Gm., so that balloon tamponade was again resorted to with control of the bleeding. Death, however, occurred from liver failure. Blood transfusions were used on him extravagantly since he received a total of fifty-five in the last fifty-two days of his life, a total of 27,500 c.c. of blood. Despite these heroic measures and the untold amount of care from doctors and nurses, he actually died from the results of acute and chronic blood loss and a failing liver.

The finding at post-mortem examination of one small opening (Fig. 1) in an esophageal varix no larger than the diameter of a match was of extreme importance, since it was through this small opening that this man's lifeblood escaped. It was decided, therefore, that in future cases of a similar nature a direct attack should be made on the bleeding varix at the time of acute hemorrhage, to control it by suture or ligation. Not only would this mean the saving of a life, if it were successful, but also untold quantities of blood transfusions would be conserved for other patients.

It was shortly after our patient was treated that Crile[5] published his experiences with transesophageal suture of esophageal varices* in seven cases with postsplenectomy bleeding with an extrahepatic type of portal bed block. He recommended it as a definitive procedure in these cases whereas it is our opinion that, in addition, it should be used as the first stage of a two-stage operative plan to treat especially the cirrhotic patient with bleeding esophageal varices that do not stop with conservative treatment within twelve to twenty-four hours, and in whom balloon tamponade is necessary to control the bleeding. Then at a later date, preferably within six to eight weeks if the patient's condition warrants it, the second stage should be performed; that should consist of a splenectomy and a splenorenal shunt or a direct portal vein-to-vena cava anastomosis.

METHOD OF TREATMENT

The differential diagnosis between esophageal varices and peptic ulcer at the time of acute hemorrhage from the upper gastrointestinal tract may be extremely difficult but of extreme importance, even with a known history of cirrhosis of the liver. The course of bleeding may be from an ulcer or the opposite may also be true. As an aid in making this differentiation, the balloon tube when used correctly may be of great value in determining the source of bleeding, since, if cardioesophageal tamponade controls it, one can be assured the diagnosis is esophageal varices, whereas if aspiration of the stomach continues to yield fresh blood, an ulceration of it or the duodenum is the most likely diagnosis. If doubt still exists as to the source of bleeding, a fluoroscopic examination of the esophagus with a thick suspension of barium sulfate should be performed even at the time of acute hemorrhage, because it is believed imperative to make the diagnosis in order that suture of the varices can be carried out with as little delay as possible if they are the cause of the bleeding. If esophageal varices are demonstrated by this roentgenographic procedure, one can be practically assured that they are the source of bleeding.

A good many cases of ruptured esophageal varices will stop bleeding spontaneously so that all do not require the radical method of control to be described here. It is believed justifiable to watch these patients for twelve to twenty-four hours to determine if they are continuing to bleed, restoring their blood pressure and hemoglobin with transfusions as necessary. If the bleeding, however, con-

*Since this paper was submitted for publication it has been drawn to our attention by Crile that Boerema,[16] a Dutch surgeon, in 1949 was the first to report the direct exposure of the esophageal varices and the interruption of them in one patient. It is believed his method is more radical than necessary since it consists of a longitudinal incision of the esophagus from the diaphragm to the arch of the aorta. The varices are interrupted with multiple suture ligatures and then obliterated by injections of a sclerosing solution between the points of interruption.

tinues or the patient shows signs of impending hemorrhagic shock, balloon tamponade should be instituted immediately to stop further hemorrhage. With the balloon in place and the bleeding controlled, preparation for the emergency operation can be deliberately carried out. This should first consist in restoring the blood volume to near normal by means of multiple transfusions. This can usually be quickly achieved once the bleeding source has been controlled. After it has been accomplished and with other transfusions available, the operation should be performed without delay.

The anesthetic should be administered by a well-qualified anesthetist. It is recommended that an endotracheal tube with an inflatable rubber cuff should be inserted after local cocainization of the pharynx and larynx before the general anesthetic is administered, because further bleeding may occur during induction with the danger of the aspiration of blood and blood clots. Since the majority of these patients are extremely ill and have severe liver disease, it is believed that cyclopropane is the safest anesthetic. The traction should be maintained on the balloon tube during the induction to prevent further bleeding, especially during the first stage of anesthesia.

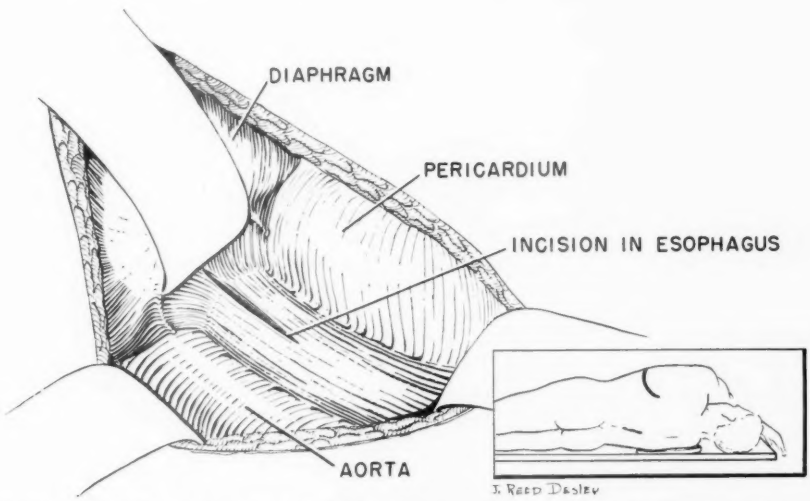

Fig. 2.—An artist's drawing to show the exposure of esophagus through a left thoracotomy incision. Note (1) partial division of diaphragm to expose the cardia of the stomach, (2) the longitudinal incision in the lower esophagus and cardia, approximately 5 cm. in length. The inset shows the position of the patient on the operating table and the location of the thoracotomy incision.

After full anesthetization, the patient is placed in a right lateral decubitus position. The left pleural space is opened by resecting the seventh or eighth rib subperiosteally. The esophagus is readily exposed by dividing the inferior pulmonary ligament. A longitudinal incision is made in the esophagus just above the diaphragm, and carried down about 2 cm. into the cardia of the stomach, first partially severing the diaphragm to expose the upper portion of the stomach. The total length of the incision need be no longer than 5 cm. since, with narrow retractors, additional exposure may be obtained both downward into the stomach and upward into the esophagus (Fig. 2). The esophageal varices usually are found to fill the entire lumen of the esophagus similar to

the appearance of large hemorrhoids in the anal canal, in some instances measuring as much as a centimeter in diameter (Fig. 3). The actual site of rupture in some cases has been visualized. The bleeding is readily controlled by pressure over the bleeding source which is usually an opening about 1 to 2 mm. in diameter in a large varix.

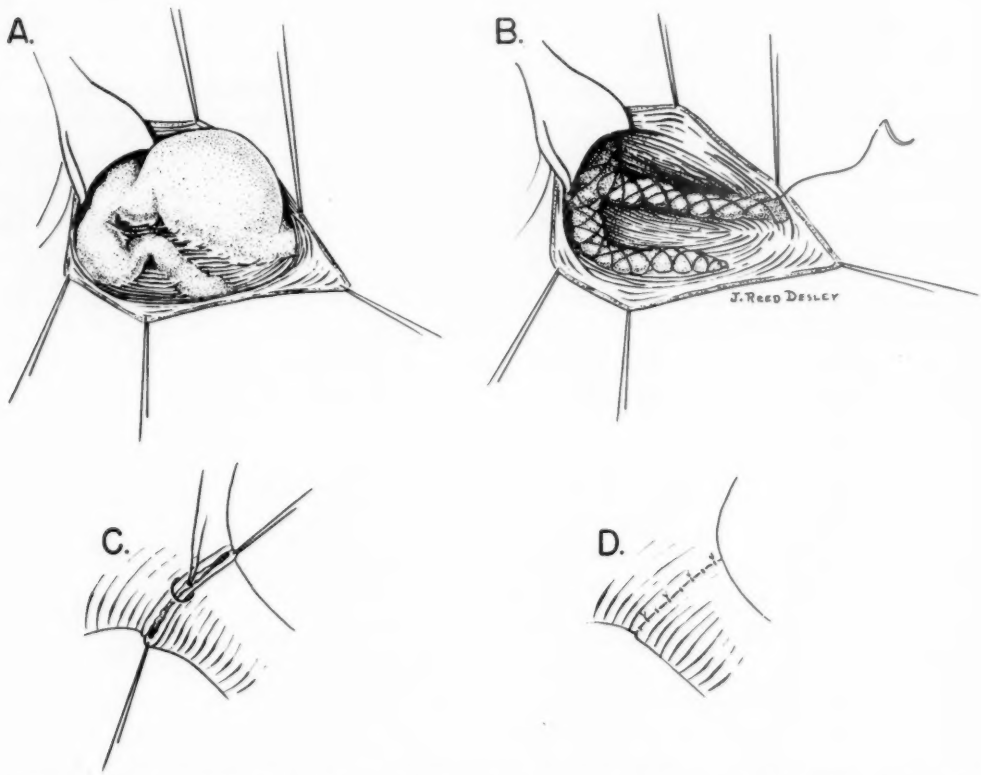

Fig. 3.—An artist's drawing to show: *A*, the huge varices in the lower end of the esophagus protruding through the incision in it; *B*, the collapsed varices after they have been sutured with No. 00 chromic catgut; *C*, the transverse closure of the longitudinal incision using interrupted fine silk sutures; *D*, the completed closure with three layers of interrupted fine silk sutures.

The method of suturing is similar to that described by Crile.[5] It consists of elevating the varix with the mucosa covering it, and suturing it with 00 chromic catgut suture on an intestinal atraumatic needle. A running over-and-over suture is used, carrying it down to and including a portion of the gastric mucosa and the varices in this region, and then running it up the esophagus for a distance of 8 to 10 cm. since, by traction on the suture as it is put in, more and more of the varices can be brought into view because of their elasticity and tortuosity. As a rule, it is possible to isolate two columns of varices as described by Crile[5] and each is sutured in this manner. In some instances it will be seen that the varices extend much higher in the esophagus than it is possible to carry the suturing, since in some cases they even extend up into the cervical esophagus.

The site of rupture in our experience, and it has been visualized in most of the cases, has been always in the esophagus within 1 to 7 cm. of the cardio-

esophageal junction, a location that is always readily included in the suturing. It is probable also if it were too high to reach, adequate obliteration of the varices below would control the bleeding because the flow of blood in these cases is always from the stomach upward. As evidence of this fact a venous pressure of 30 cm. of saline was recorded in one of the enlarged varices before suturing it, and when repeated in a varix above the area of suturing after obliteration of them, the pressure was found to be only 10 cm. of saline. Before closure of the cardioesophageal incision, the opportunity of cleaning all the old blood out of the stomach by suction and picking out the larger blood clots should be taken to aid in postoperative gastric function, and the prevention of gastric stasis. The cardioesophageal incision is made longitudinally and closed transversely in order not to reduce the lumen of the esophagus. The closure may also be done in a longitudinal manner especially if the incision has been inadvertently made too long, but care must be taken not to infold too much of the esophageal wall; otherwise a stricture of the esophagus may result. A three-layer closure has been employed, using interrupted 0000 braided No. 4 silk sutures. Primary healing of the esophageal incision without fistula formation has been obtained in all the cases, and no complications of empyema or serious infection have occurred. The thoracic incision, after suturing the esophagus, is closed in the routine manner and the pleural space is drained with a catheter which is removed in forty-eight hours. Penicillin and streptomycin are administered intramuscularly, both preoperatively and postoperatively.

RESULTS

During the past two years, transesophageal suture of bleeding esophageal varices through an esophagotomy just above the diaphragm has been performed in eleven patients as an emergency operative procedure at the time of acute hemorrhage (Table IV). There was one immediate postoperative death, or an operative mortality rate of 9 per cent. The procedure was performed in eight patients with an intrahepatic portal bed block secondary to portal cirrhosis as the first stage of a planned two-stage operative program to control the portal hypertension, and in three patients, two with an extrahepatic portal bed block and one due to cirrhosis, as a definitive procedure to control the bleeding esophageal varices as recommended by Crile.[5] Two of these latter patients were postsplenectomy bleeders, and the third was a patient in whom splenorenal and portacaval shunts had not succeeded in preventing further hemorrhage because of venous thrombosis of the portal system which was observed at the time of construction of the shunts. It is believed a longer follow-up period on these patients will be necessary to determine the true value of the procedure but it seems worth while to ascertain if it will suffice, instead of resorting to a total gastrectomy to control the bleeding as recommended by Wangensteen.[13]

The immediate postoperative death was that of a man, aged 60 years, who was in an extremely debilitated condition and in retrospect should not have been subjected to the operation. The procedure, however, was carried out as a last resort to save his life since the balloon tube had only been partially successful in controlling the esophageal bleeding and death seemed inevitable un-

TABLE IV. TRANSESOPHAGEAL SUTURE OF BLEEDING ESOPHAGEAL VARICES: RESULTS IN ELEVEN PATIENTS WITH MASSIVE HEMATEMESIS, MASSACHUSETTS GENERAL HOSPITAL (JUNE, 1950 TO APRIL, 1952)

	PATIENT	AGE	SEX	BANTI'S SYNDROME	CIRRHOSIS	BLEEDING AFTER SUTURE	TIME OF BLEEDING AFTER SUTURE	TIME BETWEEN SUTURE AND SHUNT	POST-OPERATIVE DEATHS	COMMENTS
1.	M. M.	27	F	+	0	+	3 months	–	–	A postsplenectomy bleeder. Impossible to perform a shunt. Minor bleeding episodes at 3 and 8 months, but not as severe as before varices sutured.
2.	E. P.	27	F	+	0	0	–	–	–	A postsplenectomy bleeder. Impossible to perform a shunt. No esophageal bleeding for 24 months since suture of varices.
3.	G. N.	38	M	0	+	+	5 months	–	–	Splenorenal and portacaval shunts performed but due to pre-existing portal venous thrombosis neither satisfactory. Alive and well 7 months postsuture. Bleeding at 5 months not major.
4.	A. F.	43	M	0	+	+	8 weeks	11 weeks	–	Varices sutured as first stage procedure and followed by shunt. Bleeding at 8 weeks major, requiring multiple transfusions but not as severe as before suturing of varices. No bleeding 9 months postshunt.
5.	N. S.	62	M	0	+	0	–	14 weeks	–	Varices sutured as first stage procedure and followed by shunt. No bleeding 10 months postshunt.
6.	N. P.	68	F	0	+	0	–	–	–	Varices sutured as first stage procedure, but patient died of liver failure 6 weeks postoperatively. No bleeding after suturing varices. Operative procedure did not contribute to death as patient had been discharged home 3 weeks before death.
7.	A. M.	60	F	0	+	0	–	7 weeks	–	Varices sutured as first stage procedure and followed by shunt. No bleeding 6 months postshunt.
8.	P. B.	68	M	0	+	+	6 weeks	12 weeks	–	Varices sutured as first stage procedure. Slight bleeding at 6 weeks precipitating coma, but well enough for shunt in 6 more weeks. No bleeding 6 months postshunt.
9.	G. D.	43	M	0	+	+	4 weeks	16 weeks	–	Varices sutured as first stage procedure. Bleeding and ascites at 4 weeks so shunt delayed for three months. Well and no bleeding 1 month postshunt.
10.	D. J.	60	M	0	+	–	–	–	+	Died 4 hours after varices sutured. Performed as a last resort to save his life since balloon tamponade had not controlled the bleeding.
11.	M. B.	43	F	0	+	0	–	8 weeks	–	Varices sutured as first stage procedure and followed by shunt. No bleeding 1 month postshunt.

less the hemorrhage was stopped. Another patient, a woman aged 68 years, with severe portal cirrhosis, survived the operative procedure for six weeks but then died from liver failure. It is believed the operation did not contribute to her death since she was discharged from the hospital one month postoperatively, apparently progressing satisfactorily, only to return five days later with impending liver failure. There was no further bleeding in this case after the varices were sutured. In the remaining six patients all of whom survived, there were four men, aged 43, 43, 60, and 68 years, and two women, aged 43 and 60 years. A splenectomy and an end-to-side splenorenal shunt were performed in each of these cases at periods varying from seven, eight, eleven, twelve, fourteen and sixteen weeks after the suture of the varices.

When this two-stage operative program was commenced, it was planned to take three to four months to prepare the patient for shunt surgery, but it soon became apparent that this period was too long because three of the patients had recurrent bleeding from varices, one in four weeks, another in six weeks, and the third in eight weeks. Fortunately the bleeding was not as massive as it had been in each case prior to the suture of the varices, because it ceased within a few days after a relatively few transfusions in these patients. It was then possible to take additional time to build up these patients before subjecting them to the splenorenal shunt procedure. In one patient this was performed after three weeks, in the second after six weeks, and in the third after twelve weeks. These relatively long additional periods of preparation were justified, because the bleeding stopped relatively soon in each patient and did not recur, so that it was possible to prepare them for shunt surgery without being forced to perform it before their maximum improvement had been obtained.

There were three patients who did not bleed following the suture of the varices. Splenorenal shunts were performed, respectively, at seven, eight, and fourteen weeks in these patients. At the end of these periods in each, their general condition had improved so markedly that shunt surgery was well tolerated in each case.

In view of the fact that three patients bled before shunt surgery could be performed between four and eight weeks after suturing their varices, and three did not after seven to four weeks, it is difficult to state categorically for a given case how long to wait before performing the shunt operation. At the present time it is believed imperative to wait in most cases for maximum improvement in the underlying liver disease. This should include building up the albumin level in the serum to above 3 Gm. per cent, the disappearance of the ascites, and the return of the van den Bergh test and the prothrombin time to normal or near normal levels.

The occurrence of secondary bleeding following suturing of the varices in three patients within one or two months demonstrates that the procedure should not be considered a definitive one to control the esophageal varices indefinitely and therefore should not be considered in any way a method of treatment to supplant portacaval shunts in treatment of portal hypertension either of intra-

hepatic or extrahepatic origin. At the present time it is our opinion that it is a lifesaving procedure in some patients, and in addition permits maximum preparation for shunt surgery. The following case report demonstrates these facts.

CASE 2 (M. N. S. No. 180376).—A well-developed white man, aged 62 years, was admitted to the Massachusetts General Hospital for the fifth time on May 8, 1951, at 5:00 A.M. complaining of vomiting a large amount of blood. His previous admissions had been on (1) March 4, 1932, (2) Feb. 4, 1942, (3) Feb. 8, 1948, (4) June 4, 1949. They were all because of esophagogastrointestinal bleeding arising from esophageal varices secondary to portal cirrhosis. A roentgenogram of the esophagus in 1939 revealed extensive esophageal varices. The bleeding on each admission prior to his fifth one apparently was not excessive, and ceased on conservative treatment. He refused a shunt operation on his fourth admission. It is of interest that he developed diabetes mellitus which was discovered on his third admission in 1948. At the time of his fifth admission he was very pale and obviously had lost considerable blood as his hemoglobin was 8.5 Gm. and his blood pressure 90 systolic over 60 diastolic. Blood chemistries revealed a serum protein of 6.4 Gm., albumin 3.57 Gm., and a globulin of 2.83 Gm. The prothrombin time was 62 per cent of normal. Immediate control of the esophageal bleeding was obtained with a single balloon tube and at 3:00 P.M., or ten hours after his admission to the hospital, the esophageal varices were sutured through a left thoracotomy incision. He received eight transfusions, three before operation and five during the procedure. His convalescence was uneventful and he was discharged home on May 21, 1951, the twelfth postoperative day. No further bleeding occurred and his hemoglobin was 13.5 Gm. on discharge from the hospital.

He was readmitted for the sixth time to the hospital on Aug. 14, 1951, in excellent condition, fourteen weeks after the varices had been sutured. His blood pressure was 160 systolic and 90 diastolic. Blood chemistries revealed a serum protein of 7.02 Gm., albumin 3.88 Gm., globulin 3.14 Gm. Three days later on Aug. 17, 1951, fourteen weeks after suturing the esophageal varices, a splenectomy and an end-to-side splenorenal shunt were performed under cyclopropane anesthesia. He received seven blood transfusions during the operation. His convalescence again was uneventful and he was discharged home on Sept. 5, 1951, on the nineteenth postoperative day. Since then he has remained well without further evidence of esophagogastrointestinal bleeding.

Comment.—This patient is unusual in that he has survived eleven years following his first esophageal hemorrhage despite repeated episodes since that time. His condition on the fifth admission to our hospital was more serious because of his increased age, the underlying liver disease, his diabetic condition and the fact that he had lost more blood, apparently, than on previous occasions. Because of these circumstances immediate suture of the esophageal varices was considered the best type of treatment to control the bleeding and at a later date a shunt would be performed when his condition had improved sufficiently to warrant it. In retrospect the latter procedure might have been performed sooner, especially in view of secondary bleeding in three of the other patients, but it was felt in his case, since he was a relatively old individual, it was justifiable to delay the shunt procedure providing there was evidence of continued improvement.

It is of some interest to compare the amount of blood that the patient received with this plan of therapy as compared to that of the patient in Case 1. The latter received a total of fifty-five transfusions, or 27,500 c.c. of blood, whereas the former received eight transfusions at the time of the suture of the esophageal varices and seven at the time of the splenorenal shunt, making a total of fifteen, or 7,500 c.c. of blood. In addition, this patient is alive and well whereas the other died despite these heroic measures.

SUMMARY

An attempt has been made to reduce the immediate mortality rate from bleeding esophageal varices by performing an emergency transesophageal sutur-

ing of the bleeding vessels at the time of acute hemorrhage. This procedure has been performed in eleven patients during the past two years with one immediate postoperative death, or an operative mortality of 9 per cent. It is recommended especially for the patient with cirrhosis of the liver with uncontrollable bleeding in whom balloon tamponade is necessary to stop the bleeding, since not infrequently bleeding starts again after removal of the balloon. Immediate or early suturing of the ruptured esophageal varix is considered a lifesaving procedure in these cases and it permits, in addition, adequate time to prepare the patients, many of whom are critically ill, for the major surgical procedure of constructing some type of portacaval shunt. This two-stage operative method has been accomplished successfully in six patients with bleeding esophageal varices secondary to cirrhosis of the liver.

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AN EVALUATION OF LUMBAR SYMPATHECTOMY IN TWO HUNDRED CONSECUTIVE CASES OF PERIPHERAL VASCULAR DISORDERS

JAMES A. KIRTLEY, JR., M.D., SAM Y. GARRETT, M.D., NASHVILLE, TENN., AND RAYMOND S. MARTIN, JR., M.D., JACKSON, MISS.

(From the Department of Surgery, Vanderbilt Univerity School of Medicine, and the Surgical Services, Thayer Veterans Administration Hospital and St. Thomas Hospital, Nashville, Tenn.)

DURING the past twenty-five years lumbar sympathectomy has proved to be an extremely valuable procedure with a wide range for application in the treatment of arterial insufficiency of the lower extremities. A century ago, Claude Bernard described increased blood flow following sympathetic denervation. Others slowly added to the physiologic mosaic of the sympathetic nervous system.[1] However, it was not until 1924 that surgeons began to make practical application of sympathetic denervation in disorders of the peripheral circulation.[2]

The value of sympathectomy in those disorders in which vasospasticity played a primary role was promptly accepted, but twenty years passed before its value was proved for obliterative arterial diseases in which it favored the development of collateral circulation in the smaller, unaffected vessels, though it effected no alteration in the disease process per se.[3-7]

Physiologic alterations following sympathectomy are attributed in the main to elimination of vasoconstriction. This change in the skin and subcutaneous tissues provides for an increase in surface temperature and increased tissue resistance in the extremity. Increased arterial oxygen supply to muscle assures better metabolism and in turn a decrease in the functional limitation imposed on the extremity by arterial disease. Sympathetic denervation not only reduces the pain associated with arterial insufficiency, but also eliminates the afferent pathway which contributes to reflex vasospasm. An additional beneficial effect can be attributed to the abolition of sweating, which promotes skin maceration and in cases of marked arterial insufficiency actually increases need for blood flow by increasing heat loss through evaporation.

This report is an analysis of two hundred consecutive cases followed from six months to five years in which lumbar sympathectomy was utilized in the treatment of all types of peripheral vascular disease of the lower extremity. The total number comprises ninety private patients with operations performed at Vanderbilt University and St. Thomas Hospitals by the senior author (J. A. K.), and 110 patients with operations performed at Thayer Veterans Administration Hospital, under his direction as surgical consultant during the past five years. The study was undertaken to select from this group criteria with which patients may be chosen for which the operation may be of benefit, to review the unsatisfactory results, and to note definite contraindications for sympathectomy. In this manner the clinical bases for the broadened application of the operation were reviewed and more accurate criteria were established in hopes of decreasing the number of unsatisfactory results.

Read at the sixth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 8, 1952.

VASOSPASTIC DISORDERS

Sympathectomy has proved most successful in those disorders in which vasospasm either plays the primary role or is a major factor in augmenting organic disease. Dramatic, prompt, and lasting relief may be expected if the operation is performed (1) to eliminate spasm in otherwise normal arteries, (2) to eliminate profuse sweating of an extremity, or (3) to reduce pain which either is due to tissue hypoxia secondary to arteriospasm or is a result of stimulation of afferent pain fibers which enter the central nervous system via the sympathetic nervous system. Seventy-eight patients, or 39 per cent of the group studied, were classified in this category, and, as might be expected, the result has been satisfactory in each instance (Table I).

TABLE I. LUMBAR SYMPATHECTOMY FOR VASOSPASTIC DISORDERS (78 PATIENTS)

Physiologic Results	
Elimination of spasm in otherwise normal arteries	
Elimination of profuse sweating of an extremity	
Reduction of pain due either to tissue hypoxia or stimulation of sympathetic afferent pain fibers	
Indications	
Physical injury (gunshot wound, fracture, crushing injury, arterial injury)	38
Thermal injury (burn, frostbite, immersion, or trench foot)	16
Chronic inflammation (postphlebitic extremity, chronic infection, osteomyelitis)	24

Particularly striking in this group is the large number of comparatively young persons who suffer with some degree of arterial insufficiency as the result of a vasospastic reflex initiated by the residuals of previous trauma to the extremity. Such trauma may have been a direct physical injury, as gunshot wound, fracture, crushing injury to soft parts, or direct arterial injury; thermal insult, as burn, frostbite, immersion foot, or trench foot; or the result of chronic inflammation, as follows thrombophlebitis, chronic superficial infection, or osteomyelitis. Vasospasticity resulting from trauma during younger years may not manifest clinical symptoms immediately, but can so reduce arterial supply to an extremity that symptoms of later pathologic occlusion may occur at a much younger age.

Pain, sense of constant coolness or sensitivity to exposure to cold, abnormal sweating, ulceration, and dermatitis are the most frequent presenting symptoms. The pain may be due to the diminished arterial supply or to causalgia which follows injury or perineural scarring of nerves which carry a large complement of sympathetic fibers. In either case, permanent interruption of the sympathetics results in marked and lasting relief. The resultant increase in blood flow to the extremity substitutes warmth for coolness and the vasospastic sensitivity to cold is eliminated.

Most of these patients complain of some degree of increased sweating which, in addition to its undesirable presence, sets the stage for chronic epidermophytosis, dermatitis, and ulceration. In addition to eliminating these factors, sympa-

thectomy is of great value in providing a dry surface on which to apply skin grafts with a more satisfactory degree of success.

Certainly this operation should not be advanced as a panacea for all of the sequelae of disorders exhibiting posttraumatic vasospastic reflex. The trophic ulcer in an area of hypesthesia or anesthesia is likely to continue to be a problem until pressure to the part is permanently avoided by some means. The problem of the postphlebitic extremity is a complex one, requiring judicious selection of procedures to reduce venous and lymphatic stasis by vein ligation or extirpation, and to replace ulcerated and indurated surfaces by healthier protective grafted skin. Sympathectomy may be expected to play an important role, however, by improving blood supply to the extremity and eliminating macerating perspiration.

Sympathectomy is a valuable adjunct in the management of acute arterial injuries which are complicated by inevitable vasospasm at the site of and distal to the injury. It is also useful in the improvement of collateral circulation prior to elective vascular surgery, as in the management of peripheral arterial aneurysms and arteriovenous fistulae.

ORGANIC VASCULAR DISEASES

Though sympathectomy may not be expected to halt or alter an organic disease process in peripheral arteries, it has become generally accepted that the elimination of any associated vasospasm in the smaller collateral vessels may have a profound influence in diminishing the ultimate complications due to obliterative disease. Many limbs and even lives may be preserved through its judicious use by prevention of the onset or spread of gangrene, distressing pain or claudication may be ameliorated, and persons otherwise consigned to bed or wheel chair existence may be rehabilitated. As emphasized by Primble and associates[7] the operation is *not* proposed as a frontal approach to alter organic changes already present in the vessels, but can be a flanking movement to halt accompanying vasoconstriction in the extremity.

With this thought foremost in mind we have sought to evaluate our patients who presented organic disease problems and to establish criteria by which improvement might be anticipated preoperatively.

In general our preoperative study prior to sympathectomy for organic disease, in addition to a thorough general medical evaluation, has included:

1. Critical evaluation of the extremity with regard to peripheral pulsations; angle of circulatory insufficiency; venous filling on dependency; color in positions of elevation, level, and dependency; objective gross temperature abnormalities; trophic changes, ulcerations, and extent of gangrene

2. Oscillometric observations

3. Ergometry and alteration of symptoms following paravertebral block, and skin temperature response to block or spinal anesthesia

4. Arteriography performed in selected cases, in which there was a suggestion of arterial thrombosis or segmental obstruction.

The value of preoperative skin temperature testing has been a subject of considerable debate. We have joined others in the cessation of use of the vaso-

dilator drugs in this regard because of the unpredictable generalized systemic response. A well-performed paravertebral block, of course, is the ideal means by which this test can be made, but so often results are equivocal. A low spinal anesthetic adds the disadvantage of motor paralysis and the hazard that a change in blood pressure so alters peripheral blood flow as to make the examination worthless.

Eighty per cent of patients in whom the skin temperature failed to rise preoperatively later derived a very remarkable benefit from the operation. In this regard, we postulated that the maximal amount of collateral arterial supply may not be determined by a single block or immediately following surgery, and is most probably the result of progressive dilatation of smaller vessels over a period of months. To substantiate this, we found that many of these patients noted the peak of their subjective improvement six to twelve months after operation. No significant correlation was noted between preoperative symptoms and the interval before subjective improvement. Conversely, we have also observed that most patients who have ultimately been classified as having had an unsatisfactory result failed to show any significant change in skin temperature following block preoperatively.

A second factor open to current controversy has been whether or not a surgeon is justified, in the absence of late complicating skin changes, in performing a sympathectomy for claudication or rest pain. Experimental evidence has been offered to support the contention that vasodilatation following sympathectomy for occlusive arterial disease is limited to the vessels of the skin and subcutaneous tissues and the denervation has little, if any, effect in increasing blood flow to the underlying muscles.[8] It is believed that the sympathetic nerves modulate and only partially maintain vascular tone, and smooth muscle of blood vessels are able to maintain an independent tone, thereby limiting the effectiveness of sympathectomy. This tone has been observed to be much greater in the upper than in the lower extremity.[9] Though it is difficult to refute well-controlled laboratory experiments, our clinical evidence has revealed that fifty-one of fifty-three patients who had sympathectomy performed for claudication and/or rest pain in the absence of other signs of complication of peripheral arteriosclerosis were very definitely improved. Moderation to total relief of rest pain and marked increase of exercise tolerance permitted some previously incapacitated individuals to return to gainful occupations. We have assumed that this improvement is due to an increase in muscle blood flow. In addition, it may be presumed that any patient with marked claudication or rest pain with absence of peripheral pulsations is a candidate for the early superficial complication of gangrene, and that a sympathectomy at this time may be invaluable in averting this future complication before it starts. It serves as a definite means of increasing skin resistance to the minor traumas or infections which can rapidly lead to widespread necrosis.

THROMBOANGIITIS OBLITERANS

Twenty-two patients had lumbar sympathectomy in the management of thromboangiitis obliterans. Bilateral procedures were performed on sixteen patients. Five were treated during an acute exacerbation of symptoms with

ulceration or early gangrene, while the other seventeen had the operation as an adjunct in the management of the chronic disease process. All were observed to have moderate to marked improvement immediately following surgery, as evidenced by arrest of gangrene, healing of ulceration, relief of pain, improvement of color of the foot, and/or an increase in the oscillometric index. Within periods of one to six months, however, four patients required below-knee amputations, rendering an unsatisfactory result in 18 per cent. Three of these four patients persisted in smoking after operation, despite repeated warnings.

As in arteriosclerosis, sympathetic denervation is performed to improve arterial supply to the extremity via uninvolved collaterals, and is not expected to alter the process or the progress of the arteritis. In this group of young wage earners, followed one to five years after operation, we have been impressed with the salvage or rehabilitation rate attributable to sympathectomy plus intensive conservative management.

All but two of the 82 per cent classed as satisfactory are now gainfully employed.

ARTERIOSCLEROSIS OBLITERANS

Sympathectomy was utilized in the management of one hundred patients with complications of marked arterial insufficiency of extremities due to arteriosclerosis (Table II). These patients presented one or more of the following

TABLE II. LUMBAR SYMPATHECTOMY FOR ARTERIOSCLEROSIS OBLITERANS (100 PATIENTS)

Indications: Intermittent claudication, rest pain, sensitivity to cold, impaired arterial pulsations, postural color changes, objective decreased skin temperature, trophic changes of scarring, skin, muscle, and bone, atrophy, ulceration, and gangrene. Adjunct to embolectomy	
Criteria for Improvement: Amelioration of rest pain, improvement of claudication or ulceration, arrest of gangrene, no subsequent development of gangrene	
Satisfactory Results: 76 per cent	
Unsatisfactory Results: 24 per cent	
Failure to relieve rest pain or claudication	3
Subsequent major amputation (B-K 7, supracondylar 11)	18
Paradoxical gangrene	4
Postoperative deaths	5

symptoms and observations: intermittent claudication, rest pain, sensitivity to cold, impaired arterial pulsation, postural color changes, objective decreased superficial temperature, trophic changes of scarring, skin, muscle, and bone atrophy, ulceration, and gangrene. Seventy-six per cent of this group were observed to have had a satisfactory result following sympathectomy, with amelioration of rest pain, improvement of claudication, improvement of ulceration and arrest of gangrene. Fourteen patients, who had gangrene of one or more toes at the time sympathectomy was performed, had these removed at the time of operation or shortly thereafter. As the operation cannot be expected to return viability to already necrotic tissue, limited amputations of this sort have been accepted within the realm of a satisfactory result. In none of the patients

classified as satisfactory results, followed six months to five years, was there subsequent development of gangrene.

Thirty of the one hundred cases of arteriosclerosis were further complicated by coexistent diabetes. In general the progress of their disease was accelerated as contrasted to the nondiabetics. Eight, or one-third, of the unsatisfactory results were in diabetics, a finding which suggests that the anticipated satisfactory result may be just as great in the diabetic as in the nondiabetic, if this metabolic disorder is adequately controlled and the operation is undertaken to improve the ischemia incident to the arteriosclerosis.

Twenty-four per cent of the arteriosclerotic group have been classified as unsatisfactory results. Three patients, in whom the primary consideration for the procedure was rest pain and claudication, do not admit subjective improvement. One patient, though denying improvement of pain, has had no further complication after eighteen months; the other two patients were lost to follow-up after six months.

Eighteen patients, seven of whom had deep gangrene proximal to the toes on admission, required subsequent major amputation. It may be argued that the seven patients with already existent proximal gangrene should not have been considered candidates for the operation. Seven amputations were carried out below the knee and eleven patients required supracondylar amputation. The fact that it was possible to limit amputation to a level below the knee in seven cases and that subsequent revisions have not been necessary, suggests that sympathectomy might have a role in the limitation of amputation by improving blood supply to the leg.

We have had little experience with the use of the transmetatarsal amputation as advocated by McKittrick,[10] and from our own observations feel that the indication for this procedure falls in a very narrow range. Certainly if a patient has activity otherwise limited by an associated disorder, as cardiovascular disease, arthritis, blindness, or malignancy, the level of amputation assumes less importance. Since, in the majority of cases, however, an elderly patient can be fitted with a functional prosthetic appliance, it is desirable to preserve as much of the extremity as possible, and we no longer feel that a patient suffering with gangrene of the foot due to arterial insufficiency need necessarily be forced to have a supracondylar amputation.

PARADOXICAL GANGRENE

Four patients of the group of twenty-four unsatisfactory results developed gangrene following sympathectomy. One had an arteriogram performed on the operating table with injection of 75 per cent Iodoxyl into the common iliac artery, and was observed to have a markedly ischemic extremity immediately after surgery, with subsequent gangrene necessitating mid-thigh amputation. It is believed that the local vasospasm incident to the use of this contrast medium, rather than the sympathectomy, was the precipitating cause of thrombosis in this extremity. The other three patients developed an insidious progressive gangrene within a week following surgery, suggesting thrombosis. In two

instances supracondylar amputation was performed at three weeks and one month, respectively. The third, an elderly female diabetic, died on the tenth postoperative day in cardiac failure with uncontrolled diabetes and progressive gangrene of the extremity.

It has been postulated that peripheral capillary blood flow is reduced in some critical cases following sympathectomy, by the opening of multiple proximal small arteriovenous shunts, thereby precipitating distal thrombosis. While this may at least in part be an explanation for these untoward results in each of the three cases classified as paradoxical gangrene, there was ample evidence that poor available collateral and depression during the postoperative period were the main contributory factors.

Unfortunately there are no available preoperative signs by which with certainty we can predict this hazard following sympathectomy. Though the majority of these patients may fail to exhibit any significant preoperative response to blocking, this finding alone should not be observed as a contraindication for the operation. Many patients whose skin temperatures fail to rise preoperatively would be denied a remarkably satisfactory result. The possibility likewise exists that such critical extremities might well develop gangrene necessitating amputation if sympathectomy were not performed.

Fig. 1 (Case 1).—Thrombosis of terminal aorta.

Included in the 24 per cent unsatisfactory results were five hospital deaths, which are briefly summarized:

CASE 1 (Vanderbilt University Hospital No. 193395).—C. F. N., a 47-year-old white man who was a heavy smoker and chronic alcoholic, was admitted with gangrene of the left second toe, anesthesia to the level of the knee, and loss of motor function of the left foot, suggesting popliteal artery thrombosis. The right femoral was the only palpable peripheral artery. Skin temperature rose moderately on the right and only slightly on the left following preoperative blocks. On the fifth day, a left supracondylar amputation and bilateral lumbar sympathectomies were performed. Retroperitoneal explorations revealed nonpulsatile,

firm, sclerotic terminal aorta and left common iliac artery. There was faint pulsation in the right external iliac artery. During the immediate postoperative course, the left stump healed and the temperature of the right foot increased. On the twelfth postoperative day, thrombophlebitis of the right lower extremity developed. Heparin therapy was instituted, and was followed by tromexane until the terminal stage was obvious. Progressive gangrene of both extremities ensued, necessitating repeatedly higher amputations bilaterally (Fig. 1). The patient expired three and one-half months after sympathectomy, and at autopsy thrombosis of the aorta distal to the renal arteries was demonstrated (Fig. 2). Death was attributed to general nutritional and liver failure.

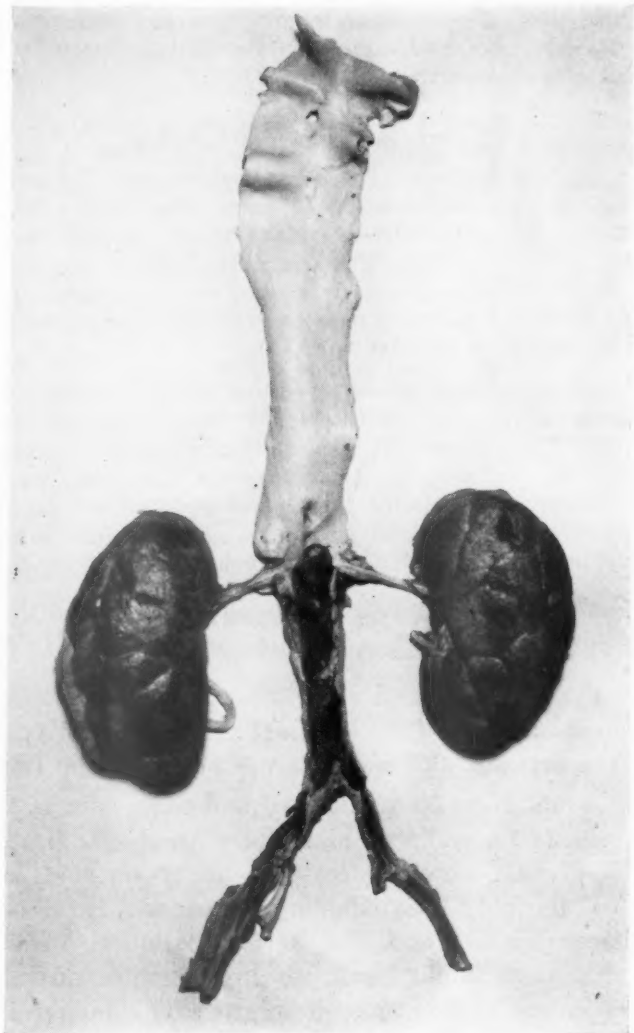

Fig. 2 (Case 1).—Autopsy specimen, thrombosis of aorta distal to renal arteries.

Case 2 (Vanderbilt University Hospital No. 152370).—M. D. N., a 63-year-old white woman, was admitted in 1946 with symptoms of claudication and coldness of both lower extremities, more marked on the right than on the left. Right lumbar sympathectomy provided excellent relief of symptoms. Two years later she returned with increased severity of symptoms on the left, and an excellent response was noted on preoperative paraspinal block. Following a relatively normal course for fours days after sympathectomy, she developed daily

temperature elevations which progressed despite vigorous antibiotic and supportive therapies. She died on the twelfth postoperative day with a terminal temperature of 108° F. During this period both lower extremities remained warm and dry. Autopsy was not obtained. Bacterial endocarditis was entertained as the cause of death because of a petechial rash and a blood culture positive for hemolytic *Staphylococcus aureus.*

CASE 3 (St. Thomas Hospital No. 8124).—O. L. B., a 60-year-old white woman with a thirteen-year history of diabetes, entered the hospital with incipient gangrene of the left foot. Three years previously she had had a right mid-thigh amputation for gangrene, and refused left lumbar sympathectomy which was advised. The operation was performed on readmission; however, the foot remained cold and pale and gangrene progressed. Cyanosis and edema of the right stump also developed, suggesting caval thrombosis. The patient lapsed into a refractory diabetic coma, and expired on the tenth postoperative day with cardiac decompensation and pulmonary edema.

CASE 4 (St. Thomas Hospital No. 7101).—L. D., a 63-year-old white woman with arteriosclerotic heart disease and auricular fibrillation, was admitted eight hours after sudden onset of severe pain and numbness of the right foot and leg. Following common femoral embolectomy, free blood flow from the distal artery was noted. Because of persistent arteriospasm, a right lumbar sympathectomy was performed. Heparin therapy was instituted four hours following surgery. Hypotension which developed during surgery remained unaltered by blood transfusion and vasopressor agents, and the patient expired fifteen hours postoperatively. At autopsy a moderate retroperitoneal hemorrhage was found, but was not believed to be the primary cause of death.

CASE 5 (St. Thomas Hospital No. 9409).—W. M. K., a 59-year-old white man, diabetic for six years, entered the hospital complaining of claudication, rest pain, cold feet, and ulceration of the right foot. Preoperative spinal block revealed a moderate increase in the temperature of the toes. Following sympathectomy the right foot remained cold and discolored. Heparin therapy was instituted on the first postoperative day; however, gangrene progressed, necessitating a supracondylar amputation on the eighth postoperative day. The following day the patient expired with a cerebrovascular accident.

THROMBOSIS OF THE TERMINAL AORTA (LERICHE'S SYNDROME)

In the series of one hundred arteriosclerotics, there were six white men who had insidious thrombosis of the terminal aorta or one or both of the common iliac arteries. The findings and results are summarized in Table III.

Ages ranged from 27 to 53 years. All had some pain in the hips or lower back, easy fatigability on walking, and intermittent claudication. Inadequate erection was noted. All were moderate to heavy smokers; only one was an alcoholic. Case 1, the only unsatisfactory result, was reviewed in the hospital death series above (Figs. 1 and 2). It is possible that the immediate and continued use of heparin might have prevented further thrombosis. The other five patients are living and have been improved by bilateral sympathectomy. Only one required amputation which was performed below the knee for a painful ulcer involving the Achilles tendon. After four years the patient manages a riding academy and rides daily.

In two cases segments of thrombosed arteries were excised at the time of sympathectomy. Both of these showed the microscopic features of arteriosclerosis obliterans, even though the last patient was only 27 years old. Leriche[12] has advocated resection of the bifurcation, but we agree with Elkin and Cooper[13]

TABLE III. INSIDIOUS THROMBOSIS OF AORTIC BIFURCATION

AGE	DURATION OF SYMPTOMS	FINDINGS ON ADMISSION	OPERATIVE PROCEDURE	EARLY RESULTS (3 MONTHS)	LATE RESULTS
1. C. N., 47	4½ years; claudication, left	Gangrene, left leg, 2 weeks	Bilateral sympathectomy; amputation, left thigh	Amputation, right leg; amputation, right thigh	Died, 3½ months
2. C. B. W., 42	6 months; left hip pain	Absent left femoral; weak dorsi flexion	Left lumbar sympathectomy	Good	Marked improvement in all symptoms, 18 months
3. R. C., 51	4 years; amputation 2 right toes; claudication, left	Absent left femoral; diabetes	Bilateral sympathectomy	Slight improvement left foot	Below knee amputation, 1½ years after operation, working daily for 4 years
4. R. T. D., 47	6 years; right hip pain; claudication	Absent right femoral; cold right foot	Bilateral sympathectomy; resection, right common iliac artery	(2½ months) right foot much warmer; hip pain improved	
5. H. A. A., 53	8 months; claudication, right; severe right hip pain	Absent right femoral	Bilateral sympathectomy	Feet hot; rest pains relieved	Claudication improved, still has some back pain, 23 months
6. Z. G. B., 27	5 years; claudication	Absent right femoral; weak left femoral; hypertension	Bilateral sympathectomy; resection, left common iliac; resection, right external iliac	Improvement of claudication; increased skin temperature on left	Gradual improvement, 4 plus years

that this is not feasible in all cases because of the marked arteriosclerosis. In retrospect, we feel that arteriectomy of the obliterated common iliac might have been of benefit in other cases, particularly the case which required amputation. Interruption of the intramural neurone reflex and other excitatory constrictor impulses further reduces vasospasm in collateral vessels.

DISCUSSION

Though we continue the use of preoperative studies to evaluate the state of progress of the disease process, we have concluded that the selection of patients for sympathectomy can be based largely on clinical findings and subjective symptoms.

This study has provided certain observations from which the following absolute and relative contraindications for surgery may be summarized. The existence of a single one of these factors may not seem justification for abstinence from sympathectomy. If either exists to a marked degree or more than one exists at least in part, they are believed to be prima facie evidence that sympathectomy is contraindicated.

Absolute Contraindications:

1. Severe cardiac, cerebral, and/or renal disease so frequently present in these patients may contraindicate anesthesia or surgical procedure.

2. Deep gangrene proximal to toes, in our experience as with others, prognosticates a poor result.

3. Rapid and progressive thrombosis of major arteries fails in most instances to be mitigated by sympathectomy.

Relative Contraindications:

4. Marked atrophy of skin and subcutaneous tissues of foot and leg with rubor of foot forecasts poor available collateral circulation.

5. Constant intractable rest pain not relieved by paraspinal or peripheral nerve blocks may not be expected to be relieved by sympathectomy.

6. Failure of basal skin temperature to respond to paravertebral or spinal block infers a relative lack of the vasospastic element complicating organic ischemia. Clinical evidence is present, however, to support the contention that smaller collateral vessels gradually dilate or overcome their local tonus to improve blood flow following release of sympathetic modulation.

After sympathectomy has been elected in the management of complications of peripheral arteriosclerosis, there are additional factors during the operative and postoperative periods which may have merit in affecting the prognosis of individual cases:

1. Judicious early use of heparin therapy in selected cases with impending gangrene may forestall the thrombosis which accounts for the majority of cases of so-called paradoxical gangrene.

2. Thromboendarterectomy, in cases of segmental occlusion, and arteriectomy, in cases of irreparable thrombosis, have been proved of value in increasing distal blood flow.

3. Gradual resumption of activity with slow walking and frequent rest, meticulous diabetic control, and careful direct instruction regarding foot hygiene are mandatory. Five of the twenty-four unsatisfactory results were attributed to problems of limited economics and poor cooperation.

4. Total abstinence from smoking is advised for all persons exhibiting arterial insufficiency. Too often this advice is ignored by the patient.

SUMMARY

1. Two hundred consecutive cases in which lumbar sympathectomies were performed have been analyzed.

2. All of seventy-eight patients who received the operation for vasospastic disorders were improved.

3. Eighty-two per cent of twenty-two victims of thromboangiitis obliterans were improved.

4. Seventy-six per cent of one hundred arteriosclerotics were improved.

5. Causes for unsatisfactory results in the remaining twenty-four arteriosclerotics were reviewed, and criteria have been suggested by which patients may be selected for sympathectomy.

6. Six cases of thrombosis of the terminal aorta and/or common iliac arteries (Leriche's syndrome) have been presented, with satisfactory results following sympathectomy in five.

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THE GROWTH OF A COLLATERAL ARTERIAL CIRCULATION IN THE SYMPATHECTOMIZED ARTERIOSCLEROTIC LEG—AN OSCILLOMETRIC STUDY

LAWRENCE N. ATLAS, M.D., LOS ANGELES, CALIF.

(From the Department of Surgery, Western Reserve University)

A MAJOR objective in the management of peripheral arteriosclerosis is the preservation of the structural integrity of the diseased limb through the development of a collateral circulation to the foot and lower leg. The fundamental mechanisms involved in the growth of a collateral arterial circulation were clarified during the latter part of the nineteenth century when Thoma[1] and Nothnagel[2] published their classic observations on the growth of collateral arterial tissue. When the circulation to a limb is reduced, the distal arteriolar bed dilates. This dilatation depends on the vasodilating action of metabolites which accumulate in ischemic tissue. Tissue deprived of blood thus becomes a region of diminished resistance to blood flow, and there is an increased flow of blood through collateral channels feeding that particular portion of the limb. The investigations of Thoma and Nothnagel demonstrated that collaterals vary in size in response to the quantity of blood flowing through them. When forced to carry an increased load over a long period of time, they hypertrophy. They increase in diameter and in length. Thus there are two phases in the development of a collateral arterial circulation: There is an early response to tissue ischemia consisting in a reduction in the peripheral resistance to blood flow with an increased flow through anastomotic channels and a subsequent reactive hypertrophy of these collaterals, a slow process of prolonged duration.

If this concept is correct, individuals afflicted with peripheral arteriosclerosis should proceed automatically to develop a collateral circulation without therapeutic assistance. Perhaps many do. Perhaps only those who fail to do so progress to the clinical stage of overt circulatory insufficiency. This failure on the part of some to spontaneously develop a collateral circulation in response to tissue ischemia can be ascribed to the antagonistic effect of sympathetic vasoconstrictor impulses on the usual vasodilator action of metabolites; and the purpose of this communication is to present oscillometric evidence of the spontaneous growth of a collateral arterial circulation following sympathetic denervation of arteriosclerotic legs.

It has always been assumed that a collateral arterial circulation does not pulsate, which is probably true of a collateral circulation which functions in response to an acute ischemic vasodilating stimulus of short duration. However, the pulsating character of a collateral circulation which functions in response to a surgically induced vasodilating stimulus of continuous and prolonged duration has not been investigated. During the past fifteen years one hundred twenty-three sympathectomized arteriosclerotic legs have been studied pre- and

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postoperatively with the oscillometer (Table I). This experience has been not only quantitatively and qualitatively significant, it has also extended over a sufficient period of time to permit the formulation of certain definite conclusions concerning the pulsating character of vascular responses to sympathetic denervation.

TABLE I. LUMBAR SYMPATHECTOMY IN PERIPHERAL ARTERIOSCLEROSIS (1937 TO 1952)

SEX	NONDIABETIC	DIABETIC
M	75	10
F	9	7

Cases: 101
Operations: 123

Note that peripheral arteriosclerosis is preponderantly a disease of men except in the diabetic.

Observation No. 1.—When chronic obliterating arterial disease is complicated by arterial spasm, sympathetic denervation, either temporary or permanent, may be followed by an immediate increase in the magnitude of measurable pulsation. This oscillometric response, which is rarely seen in peripheral arteriosclerosis, is interpreted soley on the basis of relaxation of spastic arteries. It does not signify collateral vascular growth.

CASE 1 (Diagnosis: Embolic occlusion leg artery with residual chronic reflex arterial spasm).—A man, aged 42 years, who had for many years suffered from rheumatic heart disease with auricular fibrillation, had experienced acute severe pain in the calf of his left leg six months prior to admission. Since then he had suffered from pain, coldness, and numbness of his left foot and intermittent claudication. Pulsation could not be felt in the dorsalis pedis, posterior tibial, or popliteal arteries. The femoral artery pulsated. The foot was pallid, hyperhidrotic, and cold. Following a procaine injection of the posterior tibial nerve the foot warmed rapidly to a vasodilatation level in excess of 30° C. The oscillometer recorded a trace of pulsation in the region of the lower leg just prior to surgery. Twenty-four hours following a lumbar sympathectomy the oscillometer recorded a significant increase in the magnitude of pulsation, and the popliteal artery pulsated with considerable vigor (Fig. 1).

CASE 2 (Diagnosis: Thromboangiitis obliterans complicated by arterial spasm).—A man, aged 39 years, whose chief complaint was that of repeated attacks of superficial phlebitis in his right leg, also suffered from intermittent claudication and a cold numb foot. His foot was palpably cold, cyanotic, and hyperhidrotic. There were several patches of phlebitis migrans in various stages of activity. Pulsation could not be felt distal to the common femoral artery. There was no clinically detectable vascular disease in the opposite extremity.

Fig. 2 depicts the oscillometric and skin temperature response to an intravenous injection of Priscoline. Oscillometric readings were taken at the supramalleolar levels and skin temperatures from the pads of the great toes. Following the administration of Priscoline the oscillometer recorded a measurable increase in pulsation in the ischemic limb; but pulsation in the opposite limb did not increase despite the fact that, as evidenced by the disparity in the magnitude of the digital surface temperature reactions, a greater increase in cutaneous blood flow in the normal left foot attended pharmacologic denervation.

DISCUSSION

These observations illustrate a basic principle of peripheral circulatory hemodynamics. While alterations in the volume rate of blood flow and changes in the magnitude of pulsation may be concurrent, they are actually independent

phenomena. With cardiac function constant, the volume rate of blood flow through a segment of an extremity will be determined by peripheral arteriolar resistance, while the magnitude of measurable pulsation in that same segment will be dependent on the caliber, patency, and elasticity of the arteries between the heart and the oscillometric cuff. Interrupting the flow of vasoconstricting impulses to narrowed spastic arteries relaxes their splinted walls, restores their caliber, patency, and elasticity, and enables them to transmit the pulse wave.

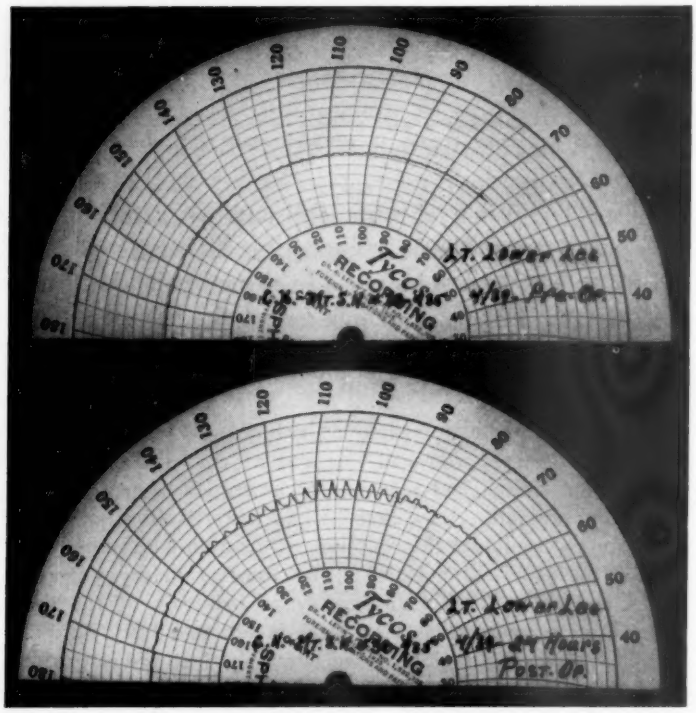

Fig. 1 (Case 1).—Oscillometric tracings recorded at the supramalleolar level of the left leg immediately before and twenty-four hours after lumbar sympathectomy.

Observation No. 2.—When there is unequivocal instrumental and clinical evidence of a collateral vascular reserve, sympathetic denervation of an arteriosclerotic leg will be followed by a *slow* and *progressive* increase in the magnitude of measurable pulsation which reaches its maximum in from six to twelve months.

Case 3 (Diagnosis: Peripheral arteriosclerosis).—A nondiabetic, 61-year-old man suffered from intermittent claudication and a cold numb foot for a period of two years. Pulsation could not be detected in the dorsalis pedis, posterior tibial, or popliteal arteries. The femoral artery pulsated. The foot was palpably cold but not cyanotic or hyperhidrotic. There was no dependent rubor. The skin of the foot was pallid but exhibited a good reactive arterial hyperemic blush. Following a paravertebral procaine injection of the lumbar sympathetic trunk digital skin temperatures rose within thirty minutes to vasodilatation levels of 31 to 32° C., and the venous filling time was eight seconds, but the oscillometer registered no pulsation at the supramalleolar level. The oscillometric response to lumbar sympathectomy is grapically portrayed in the series of tracings comprising Fig. 3. They portray a slowly progressive increase in the magnitude of pulsation which reaches its maximum nine months from the time of operation.

DISCUSSION

It is apparent that this is an entirely different pattern of pulsating vascular response than that which follows surgically induced relaxation of spastic arteries. Unless it is assumed that lumbar sympathectomy restores elasticity to sclerotic arteries, an untenable assumption, this slowly progressive increase in the magnitude of measurable pulsation can be explained only on the basis of actual hypertrophy of collateral arterial tissue.

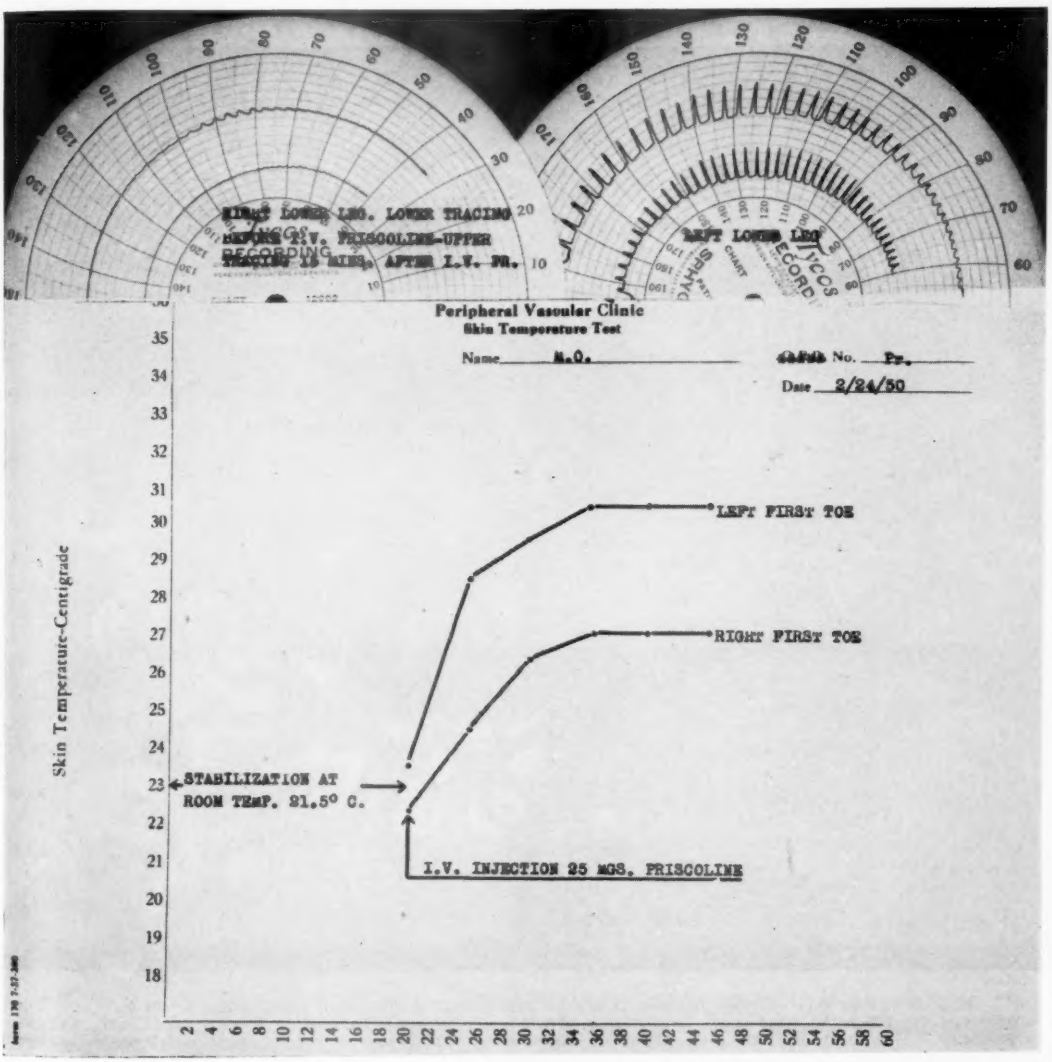

Fig. 2 (Case 2).—Oscillometric tracings recorded at the supramalleolar level of each leg immediately before and fifteen minutes after a single intravenous injection of 25 mg. of Priscoline. Thermocouple skin temperature readings taken from the pad of each great toe before and at five-minute intervals following the injection. See text for interpretation.

In 1941 the author published a preliminary report[3] of a series of cases of peripheral arteriosclerosis which had been sympathectomized, which had been observed for periods ranging from one to two years and which had demonstrated,

by virtue of the pattern of oscillometric response just described, spontaneous development of a collateral circulation. Many of these same patients have now ben continuously observed for a decade, and have enabled the author to draw at least tentative conclusions concerning the long-term value of lumbar sympathectomy in the management of selected cases of peripheral arteriosclerosis.

Conclusion No. 1.—Once the collateral circulation has attained its maximum development it will continue to function for a long period of time, and it will preserve the viability of the diseased limb.

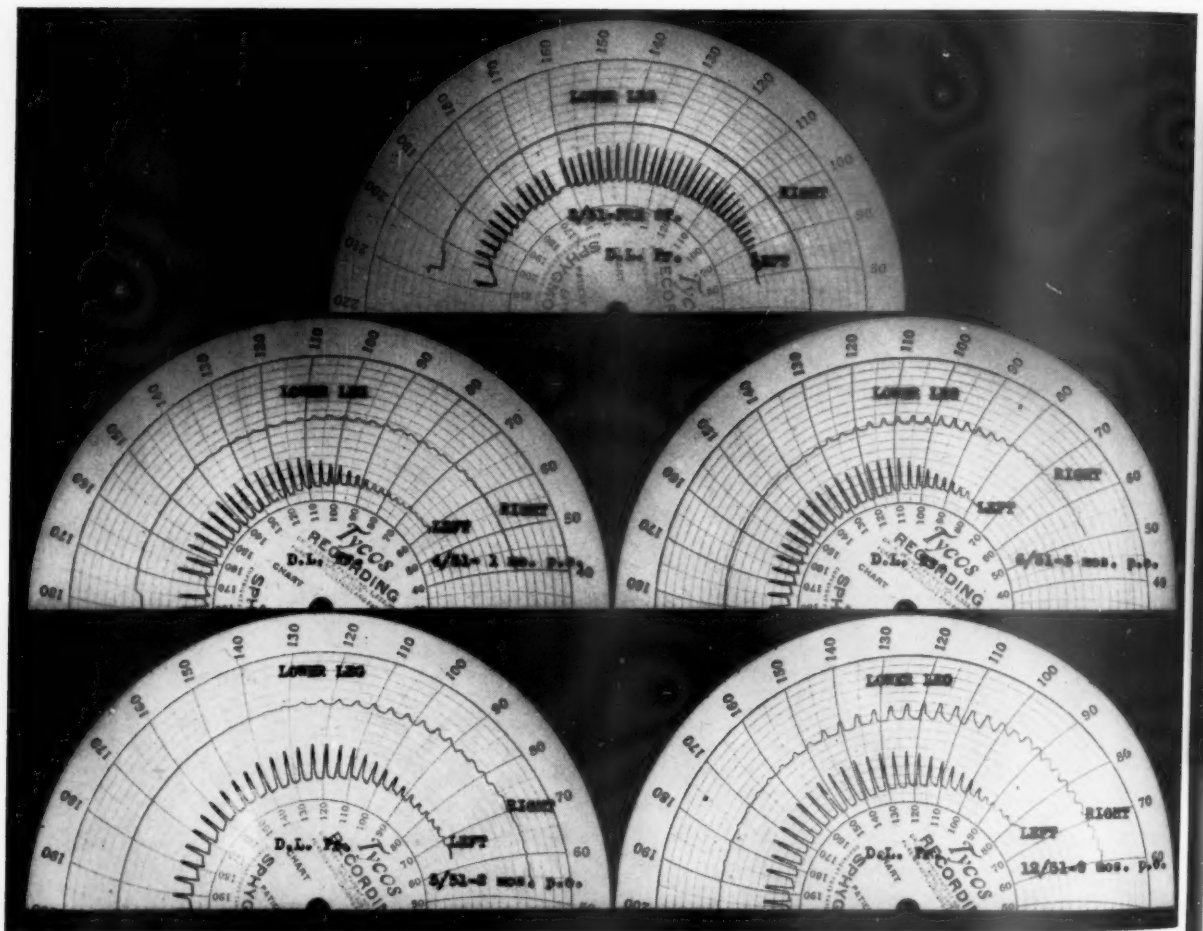

Fig. 3 (Case 3).—Oscillometric tracings recorded at the supramalleolar level of each leg immediately prior to and one, two, three, and nine months following a right lumbar sympathectomy. See text for interpretation of the slowly progressive postoperative increase in the magnitude of pulsation.

CASE 4 (Diagnosis: Peripheral arteriosclerosis).—A nondiabetic man, aged 60 years, was first observed in January of 1939. His complaint was intermittent claudication which involved the calf of the left leg. Ischemic muscle pain was accompanied by pain and numbness of the foot. His foot was always cold. Pulsation could not be felt in the dorsalis pedis, posterior tibial, or popliteal arteries. His femoral artery pulsated. His foot was palpably cold but not cyanotic or hyperhidrotic. There was no dependent rubor. The skin was pallid, but following a procaine block of the lumbar sympathetic trunk it became pink and digital

skin temperatures rose within thirty minutes to vasodilatation levels in excess of 30° C. The oscillometer registered a trace of pulsation at the supramalleolar level.

A lumbar sympathectomy was performed in February, 1939. During the first postoperative year the oscillometer recorded a progressive increase in arterial pulsation at the supramalleolar level. At the end of the first postoperative year oscillometric readings had reached their maximum value, and all the symptoms including intermittent claudication had disappeared.

Fig. 4 shows the oscillometric tracing recorded prior to sympathectomy and at the end of the first, third, and tenth postoperative years. There has been little change in the magnitude of recorded pulsation since the end of the first postoperative year. The patient was last examined in January, 1952. At that time the denervated extremity was asymptomatic, the foot was in an excellent state of nutrition, and digital skin temperatures ranged from 32 to 33° C.

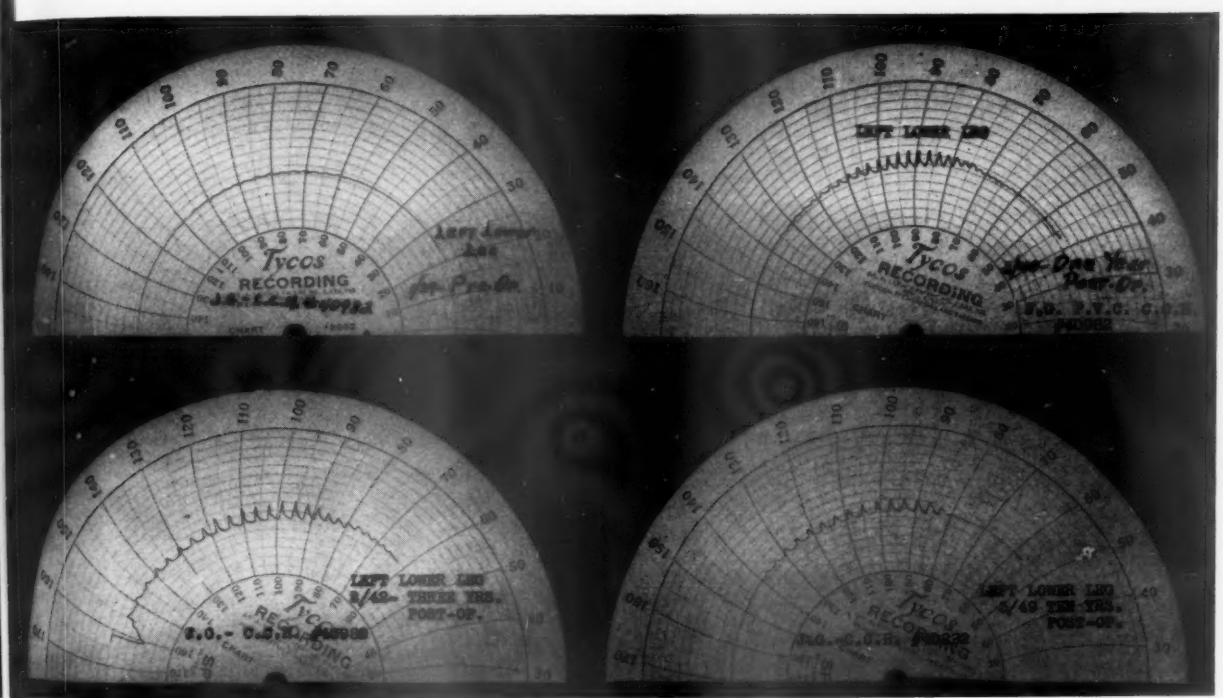

Fig. 4 (Case 4).—Oscillometric tracings recorded at the supramalleolar level of the left leg immediately prior to and one, three, and ten years following lumbar sympathectomy.

Conclusion No. 2.—Spontaneous development of a collateral circulation in the sympathectomized arteriosclerotic leg is not fortuitous circumstance.

CASE 5 (A controlled study of the therapeutic effect of sympathetic denervation of the arteriosclerotic leg).—A 56-year-old nondiabetic man, a dentist by profession, was initially observed Aug. 12, 1938. For a period of six months he had suffered from intermittent claudication. The ischemic muscle pain involved the calf of the right leg, and his right foot was subjectively cold and numb. Pulsation could not be felt in the dorsalis pedis, posterior tibial, or popliteal arteries. The foot was palpably cold but not cyanotic or hyperhidrotic. There was no dependent rubor. A roentgenogram showed calcification of the dorsalis pedis artery. The oscillometer recorded a trace of pulsation at the supramalleolar level of the right leg and an excellent degree of pulsation at the supramalleolar level of the left leg. The left dorsalis pedis and posterior tibial artery were readily palpable. However, the oscillometric reading taken at the left supramalleolar level was less than that recorded at the level

of the lower forearm which was interpreted as signifying an arteriosclerotic process in the left leg in the asymptomatic preclinical stage[4] (see Fig. 5). Following a procaine injection of the right posterior tibial nerve the skin of the sole of the foot and toes became pink and digital skin temperatures rose to values in excess of 30° C.

During the ensuing eight months the patient received intensive conservative therapy with no amelioration of symptoms or increase in oscillometric readings. Treatment consisted of intravenous hypertonic saline, Depropanex, short wave diathermy, and intermittent venous occlusion.

A right lumbar sympathectomy was performed in May, 1939. During the first postoperative year there was a progressive increase in arterial pulsation which reached its maximum at the end of twelve months. At that time the denervated limb was asymptomatic. Oscillometric tracings taken at the end of the second postoperative year are included in Fig. 5. Both extremities were asymptomatic at that time.

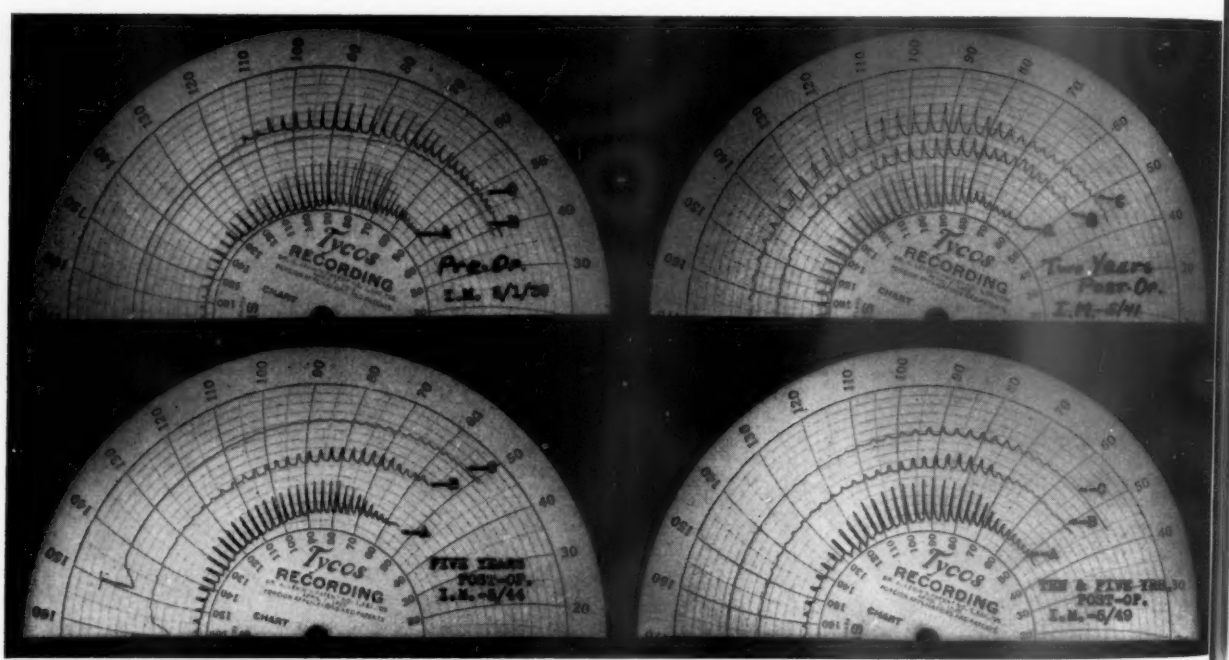

Fig. 5 (Case 5).—Oscillometric tracings recorded at the level of the right lower forearm (A), the supramalleolar level of the right leg (B), and the supramalleolar level of the left leg (C) immediately prior to *right* lumbar sympathectomy, two years following *right* lumbar sympathectomy, five years following *right* lumbar sympathectomy and immediately prior to *left* lumbar sympathectomy, and ten and five years following right and left lumbar sympathectomy, respectively. See text for interpretation of significance of arm/leg oscillometric ratio in peripheral arteriosclerosis.

The patient voluntarily returned for further consultation at the end of the fifth postoperative year. His reason for so doing was that for the past several months he had been experiencing increasingly severe intermittent claudication in the leg which had not been sympathectomized and the foot of that leg was subjectively cold and numb. The sympathectomized extremity had remained asymptomatic. The sympathectomized foot was pink, warm, and in an excellent state of nutrition. The opposite foot was cold and pallid, and pulsation had disappeared from the dorsalis pedis, posterior tibial, and popliteal arteries in the limb. The oscillometer registered approximately the same magnitude of pulsation at the supramalleolar level of the sympathectomized leg as had been recorded at the end of the second postoperative year. However, during this same period pulsation in the opposite limb had diminished to an alarming degree! (See Fig. 5.) A left-sided lumbar sympathectomy was performed.

This individual is still living and practicing his profession. Each lower extremity has remained asymptomatic, and the feet are palpably warm and in an excellent state of nutrition. Fig. 5 shows the oscillometric tracings at the tenth and fifth postoperative years.

TABLE II. A CONTROLLED STUDY OF THE SPONTANEOUS GROWTH AND MAINTENANCE OF A COLLATERAL ARTERIAL CIRCULATION IN THE SYMPATHECTOMIZED ARTERIOSCLEROTIC LEG

PATIENT	AGE	SEX	DIABETIC	OSCILLOMETRIC READINGS		INTERVAL (YEARS)
				SYMPATHECTOMIZED LEG	OPPOSITE LEG	
I. J.*	56	M	No	Trace increased to 1½	4 decreased to ½†	5
A. B.	64	M	No	½ increased to 2	2 decreased to 0‡	10
A. G.	46	M	Yes	½ increased to 2½	5 decreased to 1†	3
L. C.	53	M	Yes	Trace increased to 1	2 decreased to ¼†	3
S. S.	58	F	Yes	0 increased to ½	1 decreased to 0‡	4
H. S.	56	F	No	Trace increased to 2	6 decreased to ½†	4

*See Case 5.
†Leg sympathectomized.
‡Leg amputated because of gangrene of foot.

The author has observed a total of six cases in which a collateral circulation spontaneously developed in a sympathectomized leg and was maintained while concomitantly the circulation in the opposite limb deteriorated to the point where that limb had to be sympathectomized or, as was required in two instances, amputated because of gangrene. Pertinent data in this series of six cases in which circulation in the opposite limb served as a scientific control are summarized in Table II.

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THE ACTIONS OF INTRA-ARTERIAL DIODRAST, THOROTRAST, AND SODIUM IODIDE ON THE PERIPHERAL PULSE VOLUME OF THE LOWER EXTREMITY

R. P. JEPSON, F.R.C.S., AND F. A. SIMEONE, M.D., CLEVELAND, OHIO

(From the Department of Surgery of Western Reserve University School of Medicine, at City Hospital)

THE complications which follow the use of intra-arterial contrast media may be divided into those due to the arterial puncture, those attributable to the irritative action of the contrast media in the limb, and finally the systemic "toxic" or "sensitivity" reactions. The latter have been reviewed by Pendergrass and associates,[14] Shumacker and Godley,[15] and Dunsmore, Scoville, and Whitcomb[6] and will not be discussed in this paper.

The most severe of the limb sequelae is that of local or massive tissue necrosis leading on occasions to the loss of an extremity. This is an infrequent complication but many authors have reported one or more cases.[5, 8, 10-12] All of the commonly used contrast media have been incriminated.[5] In many instances limb gangrene has been attributed to a vasospasm and subsequent arterial thrombosis thought to be induced by the contrast media. Thus Wagner[16] states "immediate vasospastic reactions in the injected limb are very serious and can lead to gangrene".

This investigation was undertaken in order to ascertain if a severe and persistent vasospasm did in fact occur after the use of certain contrast media in lower extremities of dog and man. Particular emphasis has been given to Diodrast, as this appears to be the most popular of the present-day contrast media. The actions of sodium iodide and Thorotrast have also been studied in the animal experiments.

METHODS

Animal Experiments.—Nine dogs whose weights ranged from five to thirteen kilograms were anesthetized by intravenous pentobarbital sodium, 0.4 c.c. per kg. of body weight. Tracheotomy and bilateral cervical vagotomy were performed routinely.

An oncometer sufficient in size to include the paw of the dog and to fit loosely at the level of the ankle was made airtight by the reflexion of a cuff of skin and subcutaneous tissue from the mid-tibial level. The lower free edge of the skin was turned back for 2 cm. over the oncometer edge and secured with thread. The raw area over the leg was covered with saline-soaked gauze. Recordings of the pressure changes within the oncometer were made on an ink-writing electroplethysmograph using Statham or Grass transducers.[3] That the nervous vasomotor control of the paw remained intact could be demonstrated by the fact that electrical stimulation of the ipsilateral sympathetic chain caused a sharp fall in the paw volume and in the size of the pulse wave (Fig. 1, *C*).

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In pilot experiments it was noted that repeated punctures of the femoral artery caused a considerable modification of the paw pulse volume. In order to avoid the need for multiple arterial punctures, a small tributary of the femoral artery, either a pudendal or a muscle branch in the mid-thigh region was cannulated with a polyethylene tube (outside diameter 0.3 mm.) so that its tip lay at the junction of the branch and the femoral artery. The tube was secured by a ligature and its free end was closed with a wooden spigot. It was found that such a tube remained patent for many hours before thrombosis occurred in its lumen.

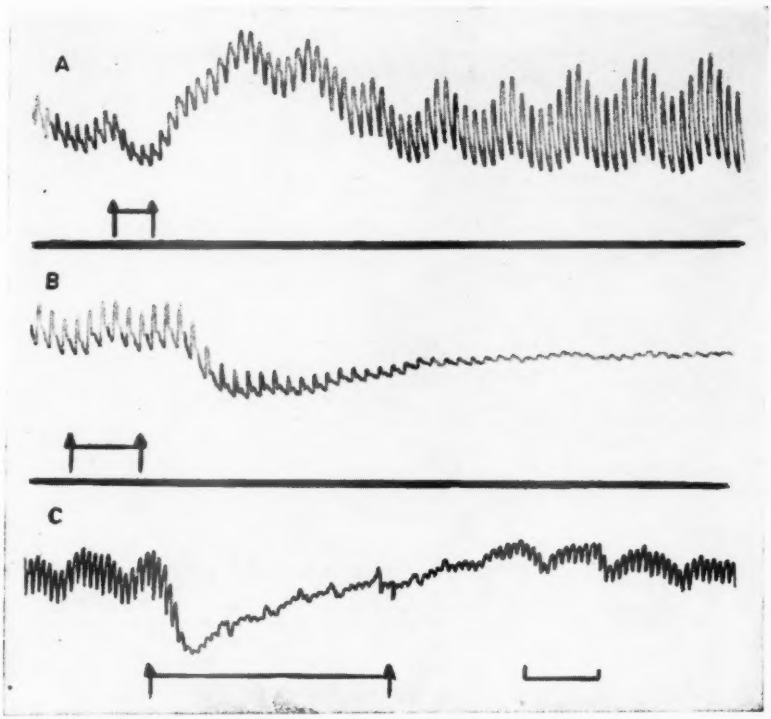

Fig. 1.—Records made by electroplethysmography from the paw of a dog. Injections into the femoral artery made between the arrows on records A and B. Record A: Injection of 0.5 mg. of acetylcholine. Record B: Injection of 2 c.c. of one in a million concentration of adrenaline. Record C: Electrical stimulation between arrows of the lumbar sympathetic chain. Time marker: four seconds.

Continuous blood pressure recordings were made from the left carotid artery in five of the animals and simultaneous readings of the pulse waves from the contralateral paw were taken in another two. Thirty-five and 70 per cent Diodrast (the diethanolamine salt of 3, 5-diiodo-4 pyridone-N-acetic acid) were injected in doses of 1 to 8 c.c., 10 per cent, sodium iodide in doses of 0.2 to 4 c.c., and Thorotrast (thorium dioxide) in doses of 1 to 2 c.c.

Tests in Man.—Records of the pulse volume were taken from the first or second toe by means of a Kal Kord sealed oncometer.[3] A transducer and a direct ink-writing electroplethysmograph were used as in the animal experiments. In one patient the percutaneous injection of Diodrast was made into the femoral artery under local anesthesia. In two others general anesthesia

was induced with intravenous pentobarbital sodium and was maintained with nitrous oxide and oxygen before the injection was made into the nonoccluded femoral artery. Patients free from or with minimal vascular disease were selected for the investigation. The dose of Diodrast injected into the femoral artery was 30 c.c. of 35 per cent solution in all cases.

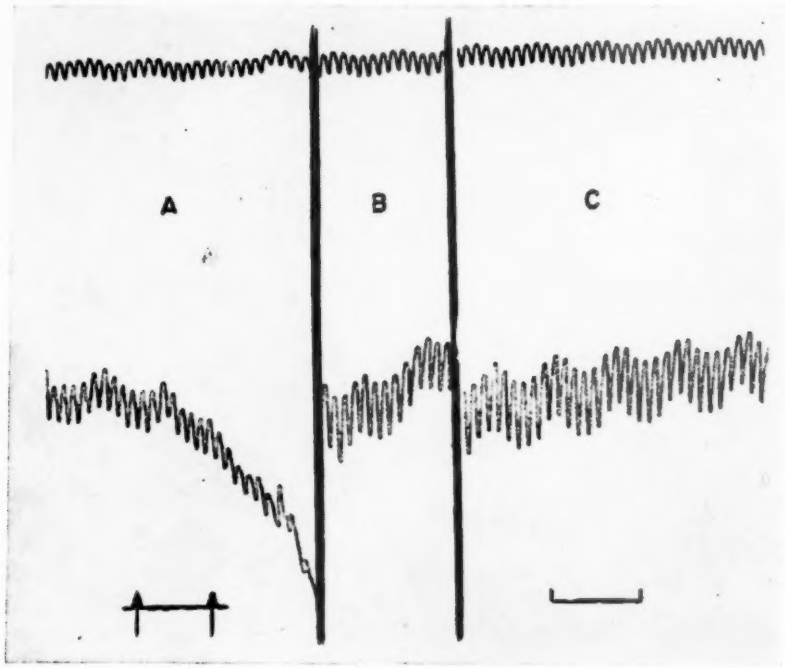

Fig. 2.—Upper records: Carotid blood pressure. Lower records made by electroplethysmography from the paw of a dog. Record A: Injection of 2 c.c. of 70 per cent Diodrast into the femoral artery between the arrows. Record B: Taken one minute after the termination of the injection. Record C: Taken two minutes after the termination of the injection. The pulse volume of the paw returned to the preinjection amplitude twenty-seven minutes after the termination of the injection. Time marker: four seconds.

RESULTS

Animal Experiments.—Three patterns of response were recorded in the pulse volume following the intra-arterial injection of Diodrast. Of the nine dogs studied, no change in pulse volume occurred in three, a vasodilatation without vasoconstriction occurred in five (Fig. 5, *D*) and an initial brief vasoconstriction was followed by a long period of vasodilatation in one (Fig. 2). The reactivity of the blood vessels to successive injections of Diodrast, although qualitatively constant in any given experiment, varied quantitatively even in the same animal. No change in the caliber of the femoral artery was observed during or after any injection.

Both the vasodilatation and vasoconstriction varied considerably in degree and persistence. Thus the vasodilatation ranged from a minimal response to one which doubled the pulse volume and which persisted for twenty-seven minutes before returning to the pre-injection level. The vasodilatation was never maximal for any animal, for it was never as great or sustained as that caused by an injection of 0.5 mg. acetylcholine (Figs. 1, *A* and 2). Similarly

the vasoconstriction never completely obliterated the pulse wave nor did it last for longer than two minutes in any of the experiments (Fig. 2). It was in no way comparable to the vasoconstriction which followed the electrical stimulation of the lumbar sympathetic chain (Fig. 1, *C*) or the intra-arterial injection of 2 c.c. of adrenaline of one in a million concentration (Fig. 1, *B*).

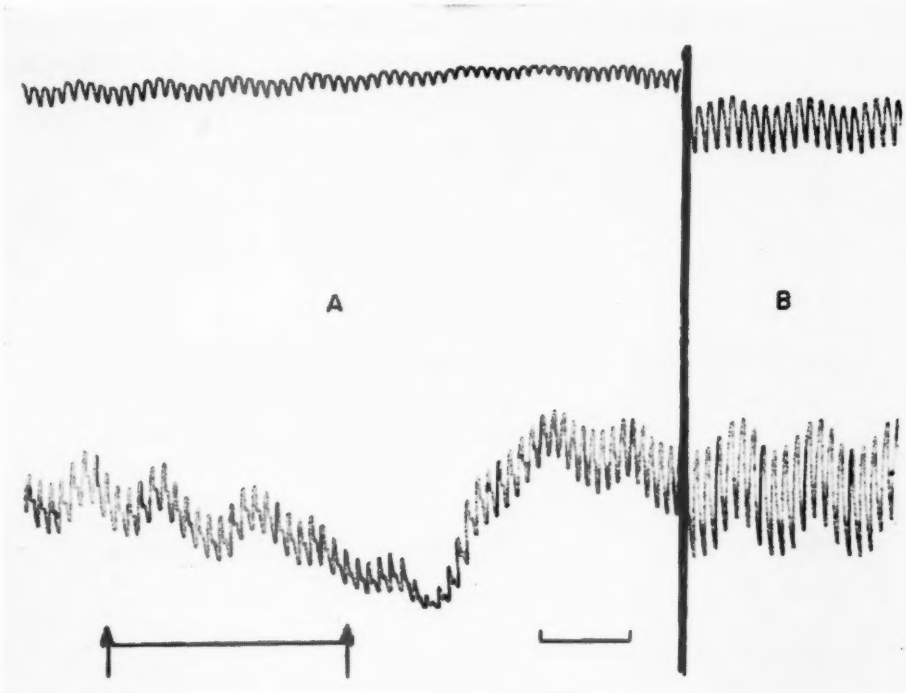

Fig. 3.—Upper records: Carotid blood pressure. Lower records made by electroplethysmography from the paw of a dog. Record A: Injection between arrows of 8 c.c. of 70 per cent Diodrast into the femoral artery. Record B: Taken thirty seconds after the termination of the injection. The amplitude of the paw pulse volume returned to the preinjection level twelve minutes after the termination of the injection. Time marker: four seconds. The increased systemic pulse volume is attributable to the decreased peripheral resistance in the limb.

The effects of intra-arterial Diodrast were studied in three dogs whose lower limbs had been sympathectomized. The lumbar sympathetic chain had been excised in one immediately prior to the injection and in the other two it had been excised for two weeks and two months, respectively. The same three types of responses were recorded, as in the animals with intact sympathetic chains (Fig. 5, *D*).

The pulse pressure recorded from the left carotid artery by means of a transducer reflected to a lesser degree the changes in the paw pulse volume (Fig. 3). In some instances where vasoconstriction was obtained in the paw, the carotid blood pressure rose immediately after the injection to 5 mm. above its previous level. The pulse rate remained unchanged. The changes in the systemic circulation are interpreted as resulting from changes in the peripheral resistance.

Injections of Diodrast (1 to 2 c.c.) into the voluntary muscle of the adductor group in the neighborhood of the femoral artery or subadventially

along the femoral artery did not change the shape or the amplitude of the pulse wave of the paw. Intra-arterial injection of Diodrast into the contralateral limb, or intravenously, gave a small increase in the pulse wave in the paw. Vasoconstriction was not recorded from such injections.

Injections of Thorotrast (1 to 2 c.c.) were made into the femoral arteries of five animals. No change in pulse wave was recorded in four of these and a vasodilatation resulted in one which persisted for about five minutes (Fig. 5, *A*). In the dog responding with vasodilatation, intra-arterial isotonic saline gave a similar response.

Fig. 4.—Upper record: carotid artery blood pressure. Lower record made by electroplethysmograph from the paw of a dog. Four cubic centimeters of 10 per cent sodium iodide injected between the arrows. Time marker: four seconds.

Injections of 0.5 to 4 c.c. of sodium iodide were studied in five dogs. In three of these dogs a vasoconstriction occurred (Fig. 4). The minimal dose to give such a response was 1 c.c. injected within three seconds. In one animal there was no alteration in the pulse volume, and in another a moderate increase in the pulse volume occurred.

Intra-arterial injections of isotonic saline (1 to 8 c.c.) were made in four dogs. An increase in the amplitude of the pulse wave was evident in two dogs (Fig. 5, *B*). This vasodilatation was present even when the saline was cooled to 15° C.

In two animals buffered phosphate solutions (2 c.c.), with pH values ranging from 5.4 to 8.0, were injected into the femoral artery. In one animal, vasodilatation resulted with all pH values (Fig. 5,*C*) while in the other animal no change of pulse amplitude resulted from any of the solutions used.

Tests in Man.—From Fig. 6 it can be seen that in one patient (Case 1) considerable vasoconstriction resulted after the injection of 30 c.c. of 35 per cent

Diodrast into the left femoral artery. Although the pulse wave did not entirely disappear, its return to preinjection height took eight minutes. In this patient the injection was made under local anesthesia and the extreme nervousness of the patient may have influenced the pulse volume return. In the remaining two patients either no diminution of the pulse volume occurred or a brief incomplete vasoconstriction from which the pulse volume recovered within two minutes. In none of the patients did an increase of the volume of the pulse wave follow the injections either immediately or later.

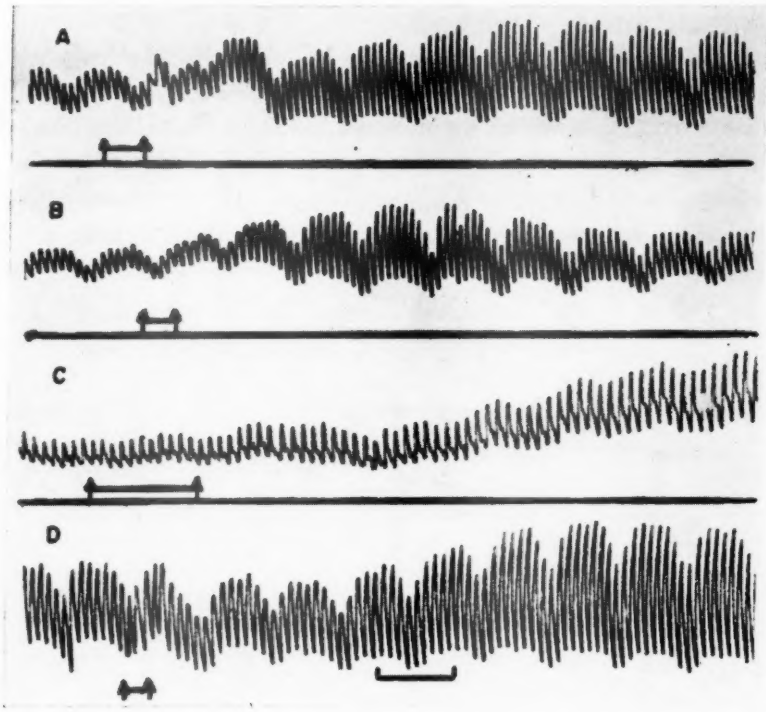

Fig. 5.—Records made by electroplethysmography from the paws of different dogs. Injections into the femoral artery made between the arrows. Record A: Injection of 2 c.c. of Thorotrast. Record B: Injection of 2 c.c. of isotonic saline. Record C: Injection of 2 c.c. of buffered phosphate solution, pH 7. Record D: Injection of 2 c.c. of 70 per cent Diodrast. A lumbar sympathectomy had been performed two weeks prior to injection D. Time marker: four seconds.

DISCUSSION

The injection of 35 or 70 per cent Diodrast into the nonoccluded femoral artery produces in the dog an increase in the size of the peripheral pulse wave preceded in some instances by a brief vasoconstriction. In human beings the vasoconstrictive phase is more prolonged and the vasodilatation is absent. It is probable, as it is in animals, that the vasoconstriction is localized in the small arteries and not in the main channels. Contiades, Naulleau, and Ungar[2] have reported similar vascular responses in dogs using a 35 per cent solution of Per-Abrodil. They stress the great individual differences between dogs and the lack of equal response with consecutive doses. These authors noted that a vasodilatation followed the intra-arterial injection of physiologic serum and hypertonic saline. Deterling[4] reported a slight elevation of the diastolic

pressures in the femoral artery tracings after intra-aortic injections of 75 per cent Neo-Iopax in both dogs and man. Foltz, Thomas, and Ward[7] have shown by direct visualization of the blood vessels of the cerebral cortex that intracarotid Diodrast causes considerable arteriolar spasm, lasting for one to two minutes. The intracarotid injection of isotonic saline did not produce these vasoconstrictive phenomena.

The vasodilatation which is seen after the injection of a wide range of innocuous solutions such as isotonic saline, serum, and buffered phosphates in both the sympathetically innervated and denervated limb, suggests that it may be due to the local effects of unphysiologic solutions upon the small peripheral vessels. The speed of injection did not correlate with the appearance of the vasodilatation. The vasoconstriction seems most likely to be produced by a direct chemical irritation of the arterial wall. Thus, those solutions containing the greatest amount of free iodine, such as sodium iodide[5] or Tenebryl[2] are the most irritating to the peripheral vasculature. Broman and Olsson[1] have demonstrated the increased permeability to dyes at the blood-brain barrier following the intracarotid injection of Diodrast and it is suggested by Paolucci[13] that all of the iodide compounds cause intimal damage in the limbs.

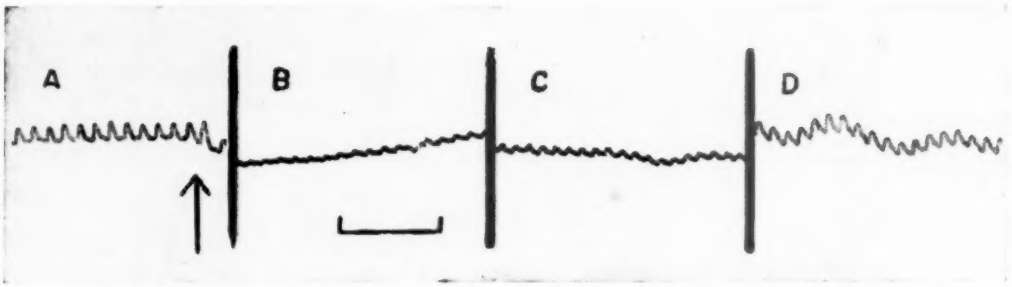

Fig. 6.—Electroplethysmographic records from the first toe of patient (Case 1). Record A: Injection of 30 c.c. of 35 per cent Diodrast commenced at arrow and completed in ten seconds. Record B: Thirty seconds after the termination of the injection. Record C: Sixty seconds after the termination of the injection. Record D: Eight minutes after the termination of the injection. Time marker: four seconds.

It is difficult to understand from the experimental evidence presented how Diodrast could invoke in the healthy limb an arterial spasm of sufficient severity and persistence to cause a major arterial thrombosis. The vasospasm which Diodrast may produce is in the medium and small-sized arteries. Large vessel spasm is difficult or impossible to provoke by chemical irritation alone, and the injection of Diodrast in the fast-flowing main arterial stream is certainly not an adequate stimulus for contracture of the main arteries. In the cases reported in the literature[5, 8, 10-12] and in those seen personally, the thrombosis leading to limb gangrene has arisen in the femoral artery. When the vessels are diseased the mural and intimal trauma of the needle puncture is probably the more important factor in the production of the thrombotic lesion. Thrombosis is promoted by the vascular spasm which may follow arterial puncture. In the upper limb it is not an unusual experience for the brachial artery to clamp down on the needle before the injection of the contrast medium has been made, and Learmonth[9] reported such an occurrence in the lower limb. The combination of intimal damage and arteriospasm by

the trauma of the needle puncture seems the most likely explanation of the occasional vascular accident, especially as the loss of a limb has been described following the injection of such a bland contrast medium as Thorotrast.[5]

SUMMARY AND CONCLUSIONS

1. Pulse volume recordings have been made with an electroplethysmograph following the injection of 35 and 70 per cent Diodrast, 10 per cent sodium iodide, and Thorotrast into the femoral artery of nine dogs. Similar recordings following the injection of 35 per cent Diodrast into the femoral artery have been made in three patients.

2. In dogs, either no change in pulse volume occurred following the injections, or a vasodilatation resulted which may be preceded by a brief vasoconstriction.

3. In human beings, the vasoconstriction phase, though not thought to be significant, was more pronounced than it was in the dog and the vasodilatation was absent.

4. From an analysis of the pulse volume recordings in dog and man it is concluded that the vasoconstriction resulting from the injection of Diodrast is insufficient to explain the occasional femoral artery thrombosis reported in the literature. It is suggested that intimal trauma and traumatic arteriospasm, especially in the diseased artery, are the more likely explanation.

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AN ANALYSIS AND FOLLOW-UP STUDY OF ONE HUNDRED POPLITEAL ANEURYSMS

RAY W. GIFFORD, JR., M.D.,* EDGAR A. HINES, JR., M.D.,* AND JOSEPH M. JANES, M.D.,** ROCHESTER, MINN.

CLINICAL experience has taught that popliteal aneurysms constantly threaten the life of the limbs that harbor them. Yet no adequate follow-up study has been carried out on patients with popliteal aneurysm to substantiate this clinical impression. Harley[1] reviewed the 35 cases of popliteal aneurysm encountered at Guy's Hospital and Linton[2] has reviewed the 42 cases of popliteal aneurysm encountered at the Massachusetts General Hospital but no attempt at follow-up was made in these cases. Furthermore only the aneurysms which are causing complications or symptoms are likely to be found in hospital records. This is evidenced by the fact that 73 per cent of Harley's patients complained of pain and 77 per cent of 22 aneurysms for which surgical extirpation or obliteration was not employed mentioned in Linton's report led to amputation of the extremity. In our series 50 per cent of the aneurysms were uncomplicated when first seen at the Mayo Clinic.

It was the purpose of this follow-up study, then, to determine whether or not the prognosis in popliteal aneurysm is grave enough to justify routine use of surgical measures designed to extirpate or obliterate the aneurysm as some authors have advocated.[2-7]

Sixty-nine patients having a total of 100 aneurysms of the popliteal artery were seen at the Mayo Clinic from 1913 to January, 1951. Aneurysms with arteriovenous communication were not included in this study. Many of the 69 patients have been seen at the clinic on one or more visits after the diagnosis of popliteal aneurysm was made at the clinic. Whenever recent information was not available, letters of inquiry were written to the patients and to their physicians. The completeness of the follow-up survey is shown in Table I.

Only 3 of the 69 patients were women. The youngest patient at time of diagnosis of the aneurysm at the clinic was 17 years old and the oldest was 88 years old. The average age at time of diagnosis was 63 years.

COEXISTING DISEASES AND SITE OF ANEURYSM

A summary of coexisting diseases of the 69 patients with popliteal aneurysms is given in Table II. The low incidence of diabetes mellitus and the high incidence of hypertension and occlusive arterial disease are noteworthy. Since most of the aneurysms were arteriosclerotic in origin, occlusive arterial disease should probably be considered another manifestation of the same disease process rather than a coexisting disease. The same is true for the presence of other aneurysms.

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*Division of Medicine, Mayo Clinic.
**Section of Orthopedic Surgery, Mayo Clinic.

TABLE I. FOLLOW-UP ON SIXTY-NINE PATIENTS WITH ONE HUNDRED POPLITEAL ANEURYSMS

EXTENT OF FOLLOW-UP DATA	PATIENTS	ANEURYSMS
Complete (from diagnosis to death or until October, 1951)	62	90
Incomplete	5	7
None	2	3
Total	69	100
Longest follow-up period:	318 months.	
Shortest follow-up period:	11 days.	
Average follow-up period:	50 months.	

TABLE II. COEXISTING OR ASSOCIATED DISEASES IN SIXTY-NINE PATIENTS WITH POPLITEAL ANEURYSM

DISEASE	PATIENTS WITH DISEASES		
	PRESENT AT TIME ANEURYSM DIAGNOSED	DEVELOPED OR DISCOVERED SUBSEQUENTLY*	TOTAL
Diabetes mellitus	1	0	1
Hypertension (more than 160/90 mm. Hg)	37	4	41
Angina pectoris or myocardial infarction	10	6	16
Cerebrovascular accident	2	8	10
Thromboangiitis obliterans	2	0	2
Arteriosclerosis obliterans	51	2	53
Syphilis (positive serology)	5	1	6
Multiple aneurysms†	32	8	40
Extra-popliteal aneurysms	11	6	17

*These are minimal figures since they may increase as time passes.
†Includes the 31 patients with bilateral popliteal aneurysms.

Forty of the 69 patients had multiple aneurysms. Thirty-one of these 40 had bilateral popliteal aneurysms and 17 patients had aneurysms in arteries other than the popliteal. The location of the 24 extra-popliteal aneurysms in these 17 patients is given in Table III. One patient, a man aged 75 years, had bilateral aneurysms of the iliac, femoral, and popliteal arteries. Six months after his initial visit to the clinic he suffered from acute arterial occlusion in the right leg and underwent a mid-thigh amputation. A longer and more detailed follow-up plus a more careful search for extra-popliteal aneurysms in this group of 69 patients would undoubtedly reveal the presence of more aneurysms.

TABLE III. SITES OF TWENTY-FOUR EXTRA-POPLITEAL ANEURYSMS IN SEVENTEEN PATIENTS WITH POPLITEAL ANEURYSMS

LOCATION OF ANEURYSM	ANEURYSMS		
	PRESENT INITIALLY	DISCOVERED SUBSEQUENTLY*	TOTAL
Abdominal aorta	4	4	8
Femoral artery	6	3	9
Iliac artery	4	0	4
Thoracic aorta	1	1	2
Dorsalis pedis artery	1	0	1

*These are minimal figures since they may increase as time passes.

Of the 100 popliteal aneurysms 46 were in the right popliteal artery and 54 were in the left.

ETIOLOGIC FACTORS

Arteriosclerosis was considered to be the cause, either entirely or in part, in 96 of the 100 aneurysms, as shown in Table IV. The clinical diagnosis of arteriosclerosis as the cause of the aneurysm was made in patients more than 40 years of age who did not have a history of significant trauma and whose serologic tests gave negative results for syphilis. There was usually, but not always, roentgenographic evidence of calcium in the wall of the aneurysm or in other arteries, or clinical evidence of occlusive arterial disease.

TABLE IV. ETIOLOGY OF THE ONE HUNDRED POPLITEAL ANEURYSMS

ETIOLOGIC FACTORS	ANEURYSMS		
	TOTAL	CLINICAL DIAGNOSIS ONLY	PATHOLOGIC DIAGNOSIS
Arteriosclerotic	92	71	21
Syphilitic	3	3	0
Arteriosclerotic and mycotic*	1	0	1
Arteriosclerotic and syphilitic	3	2	1
Traumatic	1	1	0

*Brucella cultured from wall of aneurysm.

The clinical diagnosis of syphilis as the cause of the aneurysm was made for 3 patients, ages 37, 54, and 57 years, who had positive serologic tests for syphilis and no clinical or roentgenographic evidence of arteriosclerosis.

Trauma was considered the cause of aneurysm in a 17-year-old youth who had received a gunshot wound in the popliteal area.

The clinical diagnosis of a combined syphilitic and arteriosclerotic etiology was made in 2 patients, ages 57 and 76 years, who had positive serologic tests for syphilis as well as clinical evidence of occlusive arterial disease presumably arteriosclerotic in nature.

DIAGNOSIS

Ninety-eight of the aneurysms were diagnosed clinically by palpation of a tumor behind the knee. One aneurysm was first seen on a plain roentgenogram of the thigh taken in the search for vascular calcification. One aneurysm was first diagnosed by the pathologist after amputation of the extremity for gangrene.

Roentgenograms of the popliteal area were made in 65 of the 100 aneurysms. Forty aneurysms were visualized. Twenty-nine of the aneurysms were seen on plain roentgenograms of the area. Six were visualized by arteriography and 5 were seen both on plain roentgenograms and by arteriography.

The sizes of the aneurysms varied greatly. Of those which were actually measured by the surgeon or pathologist the smallest was 3 cm. in diameter and the largest was 18 by 15 by 12 cm. By clinical estimation, the sizes varied from 2 cm. to 10 by 10 cm.

Thirty-five of the 100 aneurysms were entirely asymptomatic at the time they were first diagnosed at the clinic. Three of the 35 symptomless aneurysms were known to the patient, the others being discovered first at the

clinic. Of the 65 symptomatic aneurysms, 34 were known to the patient before coming to the clinic and 31 were first diagnosed on examination at the clinic. Of the 37 aneurysms which were known to the patient before diagnosis at the clinic, 1 had been known to be present as long as ten years, 1 had only been known for two weeks. The average time that the aneurysms had been discovered before the patients came to the clinic was seventeen months.

Symptoms associated with or related to the 65 symptomatic aneurysms had been present from one day to six years before the aneurysm was diagnosed at the clinic, with an average duration of one year. Intermittent claudication or ischemic neuritis or both occurred in association with 17 popliteal aneurysms that were not occluded by thrombosis. These symptoms could be attributed to arteriosclerosis obliterans beyond the aneurysm or to embolic showers from mural thrombi within the aneurysm. Forty-one of the symptomatic aneurysms produced symptoms which could be directly attributed to the aneurysm. These included: (1) pain in the popliteal space, (2) intermittent claudication or ischemic neuritis or both in a leg beyond an aneurysm occluded by a thrombus, (3) gangrene or ischemic ulcers secondary to thrombosis within the aneurysm or emboli from a thrombus in the aneurysm, (4) evidence of pressure on the veins or nerves in the popliteal space, (5) rupture or leaking from an aneurysm, and (6) limitation of motion of the knee due to the aneurysmal mass. Seven aneurysms produced both types of symptoms, namely, intermittent claudication and ischemic neuritis which seem related to the nonoccluded aneurysms and symptoms which can be directly attributable to the aneurysm.

COMPLICATIONS

Popliteal aneurysms have long been feared because of the frequency with which they cause complications that may lead to functional impairment if not loss of limb.

Complications were associated with 49 of the 100 popliteal aneurysms in this study when the diagnosis of aneurysm was first made at the Mayo Clinic (Table V). During the follow-up period 13 aneurysms that were originally uncomplicated became complicated and further complications developed for 10 of the originally complicated aneurysms. In all, then, 62 of the 100 aneurysms were complicated at some time in their course and a total of 121 complications occurred. The commonest complication was acute arterial occlusion with or without gangrene. The nature of the occlusion was either complete occlusion of the aneurysm by a thrombus or peripheral embolic occlusion from mural thrombi within the aneurysm. Because of the inadequacies of follow-up letters no attempt was made to distinguish between these two types of arterial occlusion when they occurred in the follow-up period. Pressure on veins in the popliteal space, rupture, and pressure on nerves in the popliteal space were the other complications listed in order of decreasing frequency. These complications led to 20 amputations, 19 of which were performed above the knee.

TABLE V. SUMMARY OF ONE HUNDRED TWENTY-ONE COMPLICATIONS OF SIXTY-TWO POPLITEAL ANEURYSMS

COMPLICATION	AT TIME OF DIAGNOSIS (49 ANEURYSMS*)	IN FOLLOW-UP PERIOD (23 ANEURYSMS*)
Thrombosis	20	0
Embolism	14	0
Thrombosis or embolism	0	11
Gangrene	10	14
Pressure on veins	19	5
Rupture	12	4
Pressure on nerves	10	2
Total	85	36

*Ten aneurysms which were complicated at time of diagnosis subsequently had further complications.

TREATMENT

The method of treatment employed in this series of 100 popliteal aneurysms is shown in Table VI. Conservative treatment usually meant no treatment at all except measures to improve the arterial circulation distal to the aneurysm if ischemia was present. Surgical measures employed were of two types: (1) procedures designed to obliterate or extirpate the aneurysm with or without previous lumbar sympathectomy and (2) amputation whenever it was necessary. Twenty-six aneurysms were removed or obliterated surgically. All but 5 of these were complicated at the time of operation. In 7 instances amputation followed conservative or no measures and was necessary at the initial visit. In 1 instance a hopelessly gangrenous foot required amputation; this was performed below the knee after successful extirpation of a popliteal aneurysm was performed to insure survival of the stump. In a sense amputation cannot be considered as surgical treatment of the aneurysm and is an admission of failure of other measures.

TABLE VI. METHOD OF TREATMENT IN ONE HUNDRED POPLITEAL ANEURYSMS AT TIME OF DIAGNOSIS AT MAYO CLINIC

METHOD OF TREATMENT	ANEURYSMS
Conservative	68
Surgical measures to extirpate or obliterate aneurysm with or without sympathectomy	21*
After trial of conservative treatment	5†
Amputation at initial visit	7
Total	101*

*One aneurysm repaired surgically to preserve below-knee amputation stump.

†Operations for three of these were performed elsewhere. Both postoperative amputations above the knee were in this group.

PROGNOSIS

The prognosis in a case of a popliteal aneurysm is greatly affected by the treatment employed (Table VII). Complications of 23 or one-third of the aneurysms conservatively treated developed in the follow-up period. These led to amputation of 11 extremities (about 1 amputation for each 6 aneurysms followed). Of the 24 limbs which survived surgical procedures on the aneurysm, 23 were followed and not one complication or amputation occurred in the follow-up period. These figures are even more impressive when it is ob-

served that the average follow-up period of the surgically treated group was more than six years while the average follow-up period of the conservatively treated group was less than four years. Twenty-one popliteal aneurysms in the conservatively treated group were followed five years or more and 16 (76 per cent) were complicated at the end of five years. If the two groups had been followed for comparable periods, therefore, the incidence of complication and amputation would be much higher in the conservatively treated group.

TABLE VII. THE PROGNOSIS IN POPLITEAL ANEURYSM; SURGICAL (EXTIRPATION OR OBLITERATION) VERSUS NONSURGICAL TREATMENT

TREATMENT	ANEURYSMS		AVERAGE FOLLOW-UP TIME (MONTHS)	PER CENT OF FOLLOWED ANEURYSMS	
	TOTAL	FOLLOWED		SUBSEQUENT COMPLICATIONS	SUBSEQUENT AMPUTATION
Conservative	73	70	44	33	16
Surgical	24*	23	74	0	0

*Does not include 2 limbs which did not survive the immediate postoperative period.

The prognosis in popliteal aneurysm not only depends on the type of treatment employed but also on whether or not the aneurysm is complicated and, if complicated, the nature of the complication.

Of the 73 aneurysms which were treated conservatively, 47 were not complicated when first diagnosed at the clinic. Forty-five of these 47 were followed for an average of forty-six months. During this follow-up period 13 (29 per cent) became complicated and the complications of 5 (11 per cent) led to amputation of the extremity. Of the 32 aneurysms which did not become complicated during the follow-up period, 24 were followed less than three years. The inference is, of course, that if the follow-up period were longer the incidence of complications and amputation would be higher.

Fifteen of the 73 aneurysms treated conservatively were completely occluded by thrombus, the limb having survived the first two months after the acute occlusive episode at time of diagnosis. Follow-up data were available concerning 14 of these 15 aneurysms. Gangrene developed in the affected limbs in 2 cases (14 per cent) in the follow-up period. Gangrene developed in 1 three months after the episode of acute thrombosis of the aneurysm and led to amputation at that time. The other patient had an ischemic ulcer on the foot six and one-half years after the acute thrombosis.

Prognosis in popliteal aneurysm is most grave when a complication other than thrombosis has occurred. For 8 (73 per cent) of 11 such aneurysms treated conservatively further complications developed in the follow-up period. These led to amputation for 5 (45 per cent) of these aneurysms in the follow-up period. Four of the 5 amputations occurred within a year of the time the aneurysm was first diagnosed at the clinic.

As said before, 20 of the 100 popliteal aneurysms in this series led to amputation of the extremities which harbored them. All but one of these

amputations were performed above the knee. Seven amputations were performed because of complications present at the time the aneurysm was first diagnosed at the clinic and no attempt was made to extirpate the aneurysm. Eleven amputations occurred during the follow-up period after conservative treatment. Thus a total of 18 amputations followed conservative treatment. This amounts to 23.4 per cent or almost 1 out of 4 of the conservatively treated aneurysms that were followed. Two amputations followed 26 surgical procedures on popliteal aneurysms, an incidence of 7.7 per cent. In both instances in which amputation followed a surgical procedure on an aneurysm, the operation had been undertaken as an emergency alternative to amputation. In one instance the rapidly enlarging aneurysm was causing severe pain with pressure on the structures in the popliteal space. In the other case the aneurysm had actually ruptured and arterial circulation distal to it had been severely compromised. Both of these aneurysms were uncomplicated until the final episode and had been seen at the clinic previously. In no instance was a surgical procedure on an aneurysm followed by amputation when the operation was performed as an elective procedure.

The complications of popliteal aneurysms that led to the 20 amputations are listed in Table VIII. Gangrene secondary to either thrombosis or embolism was by far the commonest cause for amputation in this series. In 9 instances the first complication of a previously uncomplicated aneurysm proved fatal to the limb. In 9 other instances the extremities had survived at least one previous complication of the aneurysms. The most ominous forerunning complication of this latter group was a previous episode or episodes of emoblic arterial occlusion distal to the aneurysm. This complication preceded the final complication for which amputation was performed in 6 instances.

TABLE VIII. COMPLICATIONS OF POPLITEAL ANEURYSM THAT LED TO THE TWENTY AMPUTATIONS

COMPLICATIONS OF ANEURYSM	AMPUTATIONS
Thrombosis with gangrene	8
Embolic occlusion with gangrene	3
Gangrene, presumably embolic in origin	4
Leaking or rupture	2
Undetermined	1
Gangrene following operation on aneurysm	2

Twenty-six aneurysms in 24 patients were treated surgically by extirpation or obliteration. Janes and Ivins[8] have previously reported on the results of surgical correction of popliteal aneurysm at the Mayo Clinic between 1940 and 1950. The present report includes aneurysms treated surgically before 1940 as well as those already reported in more detail by Janes and Ivins. The types of operations performed are listed in Table IX. Three of the 26 operations were performed elsewhere and the exact nature of the procedure was not determined in 2. There were no postoperative deaths.

The results of surgical extirpation or obliteration of the aneurysm are shown in Table X. The result was considered excellent in 19 cases in which

TABLE IX. SURGICAL PROCEDURES USED IN TWENTY-SIX POPLITEAL ANEURYSMS

PROCEDURE	ANEURYSMS
Lumbar sympathectomy and extirpation of aneurysm	9
Obliterative endoaneurysmorrhaphy	5
Lumbar sympathectomy and obliterative endoaneurysmorrhaphy	4
Extirpation	4*
Extirpation with amputation below knee	1
Reconstructive endoaneurysmorrhaphy	1
Undetermined	2†

*One extirpation done elsewhere.
†Operations performed elsewhere.

there was no pain, no claudication, and no edema, and the extremity was useful in every respect. Some of the patients complained of pain or edema or both in the extremity for as long as two years after the operation, but in most instances these troublesome symptoms disappeared.

TABLE X. RESULTS OF SURGICAL EXTIRPATION OR OBLITERATION OF ANEURYSMS*

RESULT	ANEURYSMS
Excellent	19
Fair	4
Amputation postoperatively	2
No follow-up information	1
Total	26

*Average time of follow-up, 74 months.

In 4 instances the result was considered as fair. Pain was still present in 2 legs operated on less than two years ago. A third patient was followed almost three years after operation and still complained of pain in the leg operated on but also had pain in the opposite leg which had not been operated on. An ulcer developed after operation on the heel of the fourth patient who was considered to have only a fair result and persisted for at least five years.

Two legs had to be amputated because of gangrene in the immediate postoperative period. The unsuccessful operations were obliterative endoaneurysmorrhaphy following lumbar sympathectomy in 1 and extirpation of the aneurysm without sympathectomy in the other. As stated previously, these 2 limbs were in such peril from rapidly enlarging aneurysms that less courageous surgeons might have chosen to amputate them without attempting any surgical attack directly on the aneurysms.

In no instance was there any complication from the aneurysm or any amputation performed in the limbs which survived the immediate postoperative period.

Complete occlusion of a popliteal aneurysm by a thrombus has been referred to as a "spontaneous cure."[3, 9, 10] Such a cure, however, is neither safe nor complete. Of the 24 aneurysms in this series which became completely occluded by a thrombus, 8 (33 per cent) were the cause of amputation of the limb within three months of the acute occlusive episode. Although none of the 16 limbs which survived three months or more after complete thrombosis

of the aneurysm had to be amputated, only 4 became symptomless, useful extremities. Intermittent claudication or ischemic neuritis or both were troublesome symptoms in 12 of the 16 surviving extremities and an ischemic ulcer of the foot occurred six and one-half years after thrombosis in 1 of these 12.

Sixty-two patients were followed from the time of diagnosis of the popliteal aneurysm at the clinic until October, 1951, or until death of the patient. In this period 32 of the 62 patients died. The causes of death are listed in Table XI. Vascular disease in one form or another was responsible for the majority of deaths. Six deaths could be attributed directly to complications of the popliteal aneurysms and in all 6 the aneurysms were in the group treated conservatively. None of the patients whose popliteal aneurysms were treated surgically died in the immediate postoperative period.

TABLE XI. CAUSES OF DEATH IN THIRTY-TWO PATIENTS WITH POPLITEAL ANEURYSM

CAUSE OF DEATH	DEATHS
Myocardial infarction	6
Rupture of abdominal aortic aneurysm	4
Carcinoma	4
Congestive heart failure	3
Postoperative death following amputation for gangrene	3*
Gangrene	3*
Cerebrovascular accident	2
Diverticulitis	2
Undetermined cause	2
Aneurysm of thoracic aorta	1
Pulmonary embolus	1
Intestinal obstruction	1

*Deaths attributable to aneurysms.

The average time of death of these 32 patients after diagnosis of the aneurysms at the clinic was forty-four months. The average age at time of death was 72 years.

COMMENT AND SUMMARY

In our series of 100 popliteal aneurysms surgical treatment by any one of a variety of procedures designed to obliterate, reconstruct, or extirpate the aneurysm carried by far the best immediate and long-range prognosis. The procedure of choice in our opinion is lumbar sympathectomy followed immediately by extirpation of the aneurysm.[8] Only one period of anesthesia should be employed. Of 21 popliteal aneurysms which were not treated by extirpation or obliteration and were followed for five years or more, only 5 were without complications at the end of five years. For 23 aneurysms which were followed for an average of six years after surgical treatment, on the other hand, there were no complications. Eighteen of the 20 amputations that had to be performed because of the complications of popliteal aneurysm were performed in the nonsurgically treated group.

A surgical procedure designed to extirpate the aneurysm with sympathectomy is the treatment of choice for all popliteal aneurysms except those occluded by thrombosis, unless the patient's general condition or life expect-

ancy militates against such treatment. It is debatable whether there is anything to gain by operating on a popliteal aneurysm which has been completely occluded by a thrombus. However, operation on a popliteal aneurysm that has caused at least one complication other than thrombosis should be undertaken as a semi-emergency procedure since the prognosis is particularly grave in this group. This is especially true if the complication is embolic arterial occlusion distal to the aneurysm.

It is not advisable to allow a patient with an uncomplicated popliteal aneurysm to wait until complications develop before surgical extirpation of the aneurysm is considered. Eleven of 20 limbs which were lost in this series of aneurysms were amputated because of complications which developed in previously uncomplicated popliteal aneurysms. Two of these 11 limbs were lost after an unsuccessful attempt to save the limb by surgical attack on the aneurysm after the complication developed.

A popliteal aneurysm is a sinister harbinger of sudden catastrophe and neither the patient nor his physician should rest easily until this threat to life and limb is surgically extirpated or obliterated.

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THE COMPREHENSIVE SURGICAL MANAGEMENT OF AORTIC SADDLE EMBOLI

JERE W. LORD, JR., M.D., AND GRAFTON BURKE, M.D., NEW YORK, N. Y.

THE occurrence of a saddle aortic embolus is one of the most catastrophic events observed in medical practice and without surgical intervention carries an immediate mortality rate of at least 75 per cent.[1, 2] When diagnosed and operated upon promptly with a satisfactory embolectomy, the chances for immediate survival are favorable. Unfortunately death from further embolic episodes and cardiac failure occurs with sufficient frequency to evoke general pessimism about the problem as a whole. Taylor[2] concluded: "Successful aortic embolectomy is so spectacular as to completely overshadow its true medical worth. It is fortunate that this procedure does not have to be considered in the light of its general medical benefit. Mankind would be far better served if the same time, effort and money were spent on some dull public health problem."

It is the purpose of this paper to present an integrated program for the management of the patient sustaining a saddle aortic embolus which includes embolectomy for the immediate problem and subsequent therapeutic measures to prevent a recurrence of another embolic episode.

PATHOGENESIS

The commonest cause of a saddle embolus is a thrombus which has formed in the auricle of the fibrillating heart. No exact data are available to shed light on the incidence of thrombosis of the appendage alone and thrombosis along the wall of the auricular cavity. Evidence presented by Beal and his associates[3] and Madden[4] suggests that in the majority of patients the appendage is the chief site for thrombus formation. In any given case there is no definitive way of establishing whether the auricular cavity contains thrombotic material along with that in the appendage. Hence the therapeutic measures to be outlined in this paper are designed, as far as possible, to control both factors.

Auricular fibrillation leading to an aortic saddle embolus is most commonly associated with mitral stenosis on the basis of an old rheumatic infection. Occasionally the cause of the fibrillation is to be found in patients with hyperthyroidism, controlled or untreated, and rarely arteriosclerotic heart disease may be the underlying pathologic lesion of the heart.

The second relatively frequent basis for a saddle embolus is a thrombus located on the wall of the left ventricle subsequent to coronary occlusion and myocardial infarction.

Rarely an aortic aneurysm or arteriosclerotic aortic plaque may lead to the casting off of a sufficiently large embolus to block the terminal aorta.

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SYMPTOMS AND SIGNS

In the literature one finds that the majority of patients sustaining a saddle aortic embolus have severe leg pain, progressing to numbness and paralysis, and then, in some, lower abdominal and low-back pain develop. In the three patients reported in this paper the outstanding initial and early symptoms were (1) severe lower abdominal, low-back, and gluteal pain and (2) paralysis of the legs. Leg pain was minimal but numbness was clearly noted by each patient. The outstanding physical signs were the absence of pulses in the femoral arteries and in the feet, and anesthesia, coldness, and pallor with or without cyanosis below the knees. In each patient there was auricular fibrillation, in two associated with mitral stenosis, and in one on the basis of a hyperthyroidism which had been successfully controlled with propylthiouracil.

TREATMENT OF THE EMBOLUS

Following prompt and accurate diagnosis, the patient should be hospitalized and the pain controlled with adequate amounts of narcotic, cardiac compensation maintained if possible, and early embolectomy performed, preferably within eight hours. We have employed spinal anesthesia, continuous in two, with the following obvious advantages: The abdominal musculature is maximally relaxed, the small bowel contracted, and the peripheral vasomotor tone abolished. Oxygen is administered to aid cardiopulmonary function and Pentothal Sodium to control apprehension. A left midrectus muscle-splitting incision centered at the umbilicus is used and the small bowel packed in towels on the anterior abdominal wall above the incision. The posterior peritoneum is incised over the terminal aorta exposing that vessel and the first two inches of each common iliac artery. Double turns of umbilical tape are placed around the three vessels and also a Blakemore rubber-shod clamp is placed in readiness around the aorta. An arteriotomy 10 to 12 mm. long is made in the right common iliac artery at its origin and the distal part of the embolus extruded and also any propagated thrombus from the distal right iliac artery. If necessary, retrograde milking of the external and common iliac arteries should be done to obtain free bleeding.

The tape is now drawn taut distal to the arteriotomy and with a right angle clamp the embolus in the left common iliac artery is removed and the same milking maneuver carried out on that side if necessary. Following tightening of the tape on the left common iliac artery, the main body of the embolus is expressed from the aorta through the arteriotomy by pressing from above. When this is accomplished, a sudden gush of blood follows and is promptly controlled by closing the jaws of the Blakemore clamp. As many authors have pointed out, digital pressure on the aorta or tightening of the umbilical tape will suffice to control bleeding, but we prefer the Blakemore clamp for its quick and relatively atraumatic control of the aorta. The distal iliacs are once again allowed to back bleed and then, after control, the arteriotomy is sutured with a continuous everting mattress suture of 00000 braided silk on an atraumatic needle. Following release of the constricting mechanism, 100 mm. of heparin is injected into the

distal aorta. The posterior peritoneum is carefully closed before closing the incision.

IMMEDIATE POSTOPERATIVE MANAGEMENT

A. Heparin.—It is in this phase of the care of the patient that widespread disagreement is noted by a number of authors. Madden,[5] for one, feels strongly that postoperative heparin therapy is contraindicated, based on two deaths due to hemorrhage following technically successful aortic embolectomy. We take the opposite point of view regarding the use of anticoagulants for the following reasons: (1) In certain instances of technically successful aortic embolectomy, full normal pulsations are not immediately present in the pedal pulses (Taylor's case, the three cases reported herein) and the presumptive reason for their absence is sludge formation or early thrombosis occurring in the small arteries in the distal parts of the lower extremities between the onset of the saddle embolus and the complete clearance of the aorta and iliac arteries. Although it is entirely possible that such clearance may take place spontaneously, we believe that the use of heparin aids in the complete return of arterial flow. (2) The literature is replete with instances of successful aortic embolectomies only to have a second or third embolus lead to death of the patient two weeks or longer after the operation. A study of Taylor's[2] table reveals that out of twenty-seven "successful" cases of aortic embolectomy, seven died from further embolic episodes and four others survived further embolic episodes. For that reason we believe that immediate heparinization is indicated and should progress into long-term anticoagulant therapy, as Foley[6] has clearly shown that adequately controlled prolonged anticoagulant therapy will eliminate the likelihood, in the vast majority of instances, of another embolus. However, as he has pointed out, no matter how long anticoagulant therapy is employed, if it is stopped, the hazard of another embolus is exactly as great as though no anticoagulant had ever been used. (3) A less important indication for anticoagulant therapy is to be found in the possible occurrence of venous thrombosis in the legs precipitated by the embolic arterial interruption. (4) The need for protection of the suture line and local trauma to the endothelium is only of value in the occasional patient in whom the time of embolectomy is delayed ten or more hours after onset.

Heparin is at one and the same time a most valuable and dangerous drug. We have employed it in these patients in the following manner: 100 mg. is injected directly into the aorta before closing the posterior peritoneum. Immediately postoperatively a clotting time has been taken and heparin administered by intermittent subcutaneous injections in two patients and a continuous intravenous drip in the third. Clotting times have been taken at intervals of three to four hours and the following day Tromexan or Dicumarol has been started. Heparin has been discontinued in forty-eight hours in two cases when the prothrombin time was in the therapeutic range. In the first two patients ideal control of the clotting time was effected but in the third patient an uncalled-for amount of heparin was given which led directly to the death of the patient on the fourth postoperative day (see Case 3). In spite of such an unfortunate

and needless error we still believe that heparin is a valuable adjunct when carefully controlled by experienced workers, but, under circumstances such as surrounded Case 3 in this report, we believe that a far wiser course would have been to omit postoperative heparinization for the first twenty-four hours following the initial injection of 50 mg. into the aorta (Case 3 had only 50 mg. injected into the aorta because 200 mg. of Depo-Heparin had been given five hours preoperatively).

B. Cardiac Management.—The cooperation and assistance of an internist fully conversant with the cardiac patient who may be or may have been in failure is of the greatest value. Oxygen, digitalis, low-salt regime, Mercuhydrin, fluid balance and anticoagulant therapy all are best handled by the cooperative efforts of internist and surgeon.

LONG-TERM MANAGEMENT

Ideally, with the patient fully recovered from the effects of the aortic embolus with normal circulation to the legs and while on anticoagulant therapy, one should undertake studies from the viewpoint of correcting the cardiac lesion which had been responsible for the embolus. In those cases with auricular fibrillation, auricular appendectomy (Madden, Beal) should be carefully considered. In patients who have, in addition to auricular fibrillation, mitral stenosis, commissurotomy combined with auricular appendectomy may well be of value (Case 2). In this way not only is cardiac function improved, but the risk of a further embolic episode is lessened. The patient in Case 2 has had the complete procedure of aortic embolectomy and commissurotomy with auricular appendectomy performed, while the patient in Case 1 is in excellent condition following aortic embolectomy and the use of long-term anticoagulant therapy without a cardiac procedure. The patient in Case 3 died four days following a satisfactory embolectomy as a result of renal failure which in turn was closely related to massive hemorrhage due to heparin overdosage.

CASE REPORTS

CASE 1.—M. McC., a 42-year-old white schoolteacher, had suffered from mitral stenosis and abnormalities of cardiac rhythm for several years. Five hours before admission to the New York Hospital on March 23, 1950, as a private patient of Dr. Irving S. Wright, she had experienced marked numbness and heaviness of both legs from the hips to the feet. Paralysis soon ensued. There was only mild pain located chiefly in the lower back and gluteal region. The femoral and pedal pulses were absent, and both legs below the knees were profoundly cold. Anesthesia was complete in the feet which were moderately cyanotic. Seven hours after onset, the saddle embolus was removed through a transperitoneal approach under continuous spinal anesthesia. Immediately postoperatively the femoral pulses were palpable and a good posterior tibial pulsation was present on the left side. The right leg was warm but the foot remained cooler than the left. One hundred milligrams of heparin was injected into the aorta before closure of the incision, and heparin was given by subcutaneous injection for twenty-four hours. Tromexan was started twelve hours postoperatively at which time a strong right posterior tibial arterial pulsation was observed.

The patient made an uneventful recovery and was discharged from the hospital on the twenty-third postoperative day. She has been on long-term anticoagulant therapy for the past twenty-six months, has been active with her housework, and has had a normal arterial circulation to the lower extremities.

CASE 2.—V. McC., a 44-year-old white telephone operator, was admitted to the New York Hospital on Jan. 25, 1951, as a private patient of Dr. William T. Foley, complaining of severe lower back and abdominal pain with milder pain in the legs and paralysis of both legs. The patient had a long-standing mitral stenosis associated with auricular fibrillation for five years and had experienced several episodes of pulmonary edema. Her cardiac status had been fairly well controlled on a low-salt diet, digitoxin, and occasional injections of Mercuhydrin. On admission, pulsations were absent in both femoral arteries and in those of the feet. The legs were cool below the mid-thigh. Seven hours after onset the saddle embolus was removed transperitoneally under continuous spinal anesthesia. One hundred milligrams of heparin was injected into the aorta before closure and heparin administered postoperatively for the first forty-eight hours when the prothrombin time reached the therapeutic range as a result of orally administered Dicumarol.

Immediately postoperatively the right femoral artery pulsated feebly but no pulse could be palpated on the left side. The following day, easily palpable pulses were present in the right foot but the left femoral artery did not pulsate until the thirteenth postoperative day and pulsations in the left foot became weakly palpable by the third postoperative month.

In view of the marked limitation of the patient's activity due to mitral stenosis, i.e., 2 pillow orthopnea, 1 flight dyspnea, and the necessarily strict cardiac regime, studies of cardiac dynamics were performed by Drs. Daniel S. Lukas and Charles T. Dotter which are shown in Table I. Since the findings also revealed low pulmonary resistance and there was no evidence of mitral insufficiency or aortic valvular disease, a mitral commissurotomy with a left auricular appendectomy was carried out on April 14, 1951. Dicumarol was discontinued three days preoperatively and Tromexan employed until twenty-four hours before operation when the prothrombin time was twenty-two seconds. Postoperatively hearin was administered subcutaneously in 30 mm. dosage every six hours until the prothrombin time reached the therapeutic range on the sixth day as a result of oral Dicumarol begun on the fourth postoperative day. Her convalescence was uneventful and cardiac catheterization studies were repeated on Sept. 21, 1951. The salient data are shown in Table I. Long-term anticoagulant therapy was discontinued in November, 1951, and the patient was in excellent condition when examined on May 9, 1952. She has worked regularly for the past twelve months, is able to climb two to three flights of stairs without dyspnea, is no longer on salt restriction, and takes digitoxin as her only medication. She has gained 14 pounds since her second operation.

TABLE I. CARDIAC CATHETERIZATION (CASE 2)

	PREOPERATIVE	POSTOPERATIVE
Cardiac output		
L./min.		
Rest	3.78	4.57
Exercise	3.73	4.95
Pulmonary artery		
Mm. Hg		
Rest	57/24	45/21
Exercise	76/39	74/34
Pulmonary capillary		
Mm. Hg		
Rest	26	18
Exercise	34	32
Mitral valve area in cm.^{2}	0.70	2.35

CASE 3.—E. T., a 56-year-old white woman, had been treated successfully for hyperthyroidism associated with auricular fibrillation by propylthiouracil. She was relatively well until 6:00 P. M. Feb. 11, 1952, when she experienced severe pain in the lower back and abdomen, buttocks, and legs. Pulsations were absent in the femoral and pedal arteries and the legs were cold below the knees. The patient was admitted to a Long Island hospital where seven hours after onset of the saddle embolus a transperitoneal embolectomy through

the right common iliac artery was carried out. Fifty milligrams of heparin was injected into the aorta before closure (only 50 mg. of heparin was injected because 200 mg. of Depo-Heparin had been given five hours preoperatively). On removal of the drapes, strong pulsations were present in the femoral arteries but no pulse could be felt in either foot.

Immediately postoperatively, the clotting time was fourteen minutes. A continuous intravenous drip of 100 mg. of heparin in 1,000 c.c. of 5 per cent glucose in distilled water was started postoperatively. It was planned to have this infusion run for eight to twelve hours, in order to maintain the clotting time at fifteen minutes. Unfortunately, the nurse allowed 750 c.c. to run in during the first three hours postoperatively. Bleeding from the wound was noted at 6:00 A.M. and the patient's incision was opened at 8:00 A.M. by her local surgeon in order to find a bleeding point. Diffuse oozing was observed and then a clotting time was drawn which subsequently proved to be two hours. Fifty milligrams of Protamine Sulfate was administered and by 11:00 A.M. the clotting time was five minutes.

Careful control of heparin therapy was then instituted during the ensuing forty-eight hours. Unfortunately renal function which had been adequate for twenty-four hours became suppressed and the patient died on the fourth postoperative day. The viability of both feet seemed likely although pulses were never felt in them during the postoperative period. Autopsy showed that the embolus had arisen from the left auricle near the posterior mitral leaflet. At this site it probably also extended into the auricular appendage. The abdomen contained approximately 2 L. of fluid and clotted blood. The site of the arteriotomy was healing cleanly without evidence of local hemorrhage. No thrombi were found in the iliac arteries.

Comment.—Inexperience with the administration of heparin and the lack of an appreciation of the danger of an overdosage led directly to the patient's death. Since this patient was operated upon in an outlying hospital where it was impossible to give personal supervision to the immediate postoperative heparin therapy, unquestionably it would have been better judgment to have withheld the administration of intravenous heparin for twenty-four hours following the operation. We have recently had the experience of starting heparin fourteen hours postoperatively in a 22-month-old child for cerebral thrombosis following a Blalock-Taussig procedure for tetralogy of Fallot. The preoperative hematocrit had been 74 and heparin was given in such amounts as to lead to a clotting time of three hours, some eight hours following the cerebral accident. Of interest is the fact that no bleeding occurred through the catheter in the chest or elsewhere and the child made a full and complete recovery. Needless to say, heparin was discontinued following the report of the incredibly long clotting time. It would seem fair to conclude that hemorrhage due to prolongation of the clotting time within the therapeutic range is not likely to occur if twelve or more hours have elapsed from the time of the operation and the institution of heparin therapy.

DISCUSSION

The comprehensive management of an aortic saddle embolus includes the following: (1) accurate diagnosis, (2) early prompt surgical procedure (within eight hours), (3) transabdominal embolectomy under spinal anesthesia, (4) postoperative heparinization precisely controlled to avoid hemorrhage, (5) careful and thorough cardiac management including close cooperation between internist and surgeon, (6) long-term anticoagulant therapy, (7) cardiac catheterization studies in those patients with mitral stenosis and auricular fibrillation to evaluate properly the possible value of commissurotomy in conjunction with auricular appendectomy, (8) auricular appendectomy with or without commissurotomy dependent on the clinical evaluation of the patient combined with data obtained under point 7. (9) In those patients whose embolus arose from a mural thrombus secondary to myocardial infarction, long-term anticoagulant

therapy should be continued for the remainder of the patient's life. Discontinuance of such therapy will invite, in the majority of instances, a recurrent embolic episode.

When the above integrated program is followed, a catastrophic event, which has an immediate mortality rate of at least 75 per cent under conservative management and a life expectancy of only a few weeks to a few months after successful embolectomy in all but a few recorded cases, can be managed successfully in the majority of patients, not only on an immediate basis but also throughout a long-term follow-up.

CONCLUSIONS

The comprehensive surgical management of saddle aortic emboli includes prompt embolectomy, long-term anticoagulant therapy, left auricular appendectomy in selected cases combined with mitral commissurotomy when indicated. Three patients operated upon through a transperitoneal approach have successfully undergone aortic embolectomy. One patient died on the fourth postoperative day because of inadequate postoperative control of heparin therapy. The other two patients are alive and well, working full time, on long-term anticoagulant therapy in one patient, and after mitral commissurotomy and left auricular appendectomy in the other. We believe that the integrated surgical program outlined in this paper should lead to a more optimistic view of a lesion which is highly fatal when treated by means other than surgical.

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CONGENITAL DIVERTICULUM OF THE LEFT VENTRICLE

Case Report

Willis J. Potts, M.D., Arthur DeBoer, M.D., and Frank R. Johnson, M.D.,
Chicago, Ill.

(From the Department of Surgery, Children's Memorial Hospital)

RESECTION of a congenital diverticulum of the heart is still a very rare surgical procedure. We believe this case, the fourth reported, is not only that of the oldest living patient recorded but is also that of the first such patient to be operated upon in this country.

Skapinker[1] from South Africa recently reported successful removal of a diverticulum in a 2-month-old native Bantu baby and reviewed the pertinent medical literature. His patient was the third to undergo surgical correction of a diverticulum of the ventricle. Wieting,[2] the first to attack this problem in 1912, simply replaced the diverticulum and closed the opening in the diaphragm in a 3-year-old child. Follow-up studies of this patient were not reported. Roessler,[3] in 1944, successfully resected a diverticulum in a newborn child. Although surgery was successful the child subsequently showed signs of other congenital heart defects. No further information is available. The patient reported by Skapinker in 1951 was in good health eight weeks after the operation. He did not close the associated diaphragmatic defect.

The patients in the nine reported cases not corrected by surgery all died in infancy except one child who lived until she was four years of age, when she died of spontaneous rupture of the diverticulum (Von Sydow[4]).

CASE REPORT

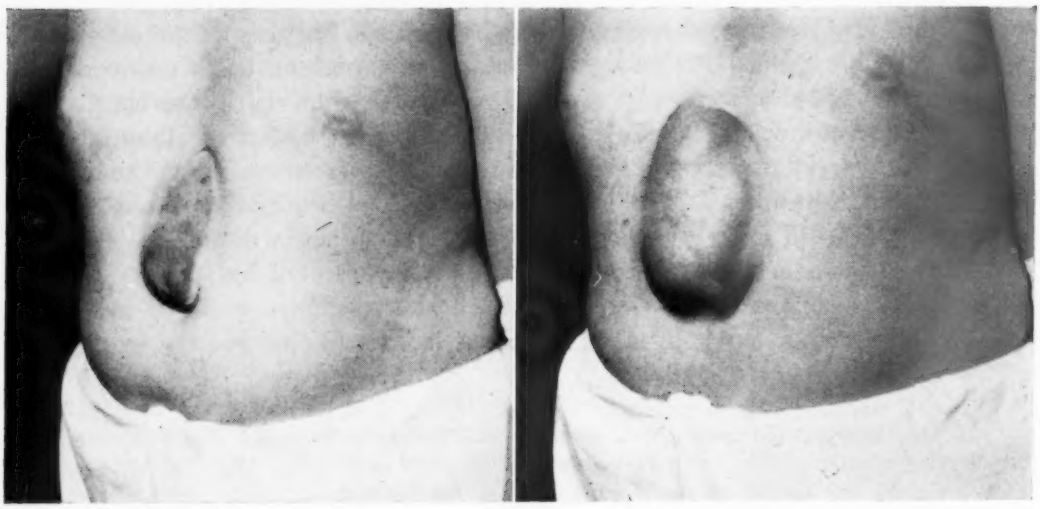
L. H., a 9-year-old male child, entered the Children's Memorial Hospital because of a pulsating mass in a large ventral hernia present since birth (Fig. 1). The mother stated that the child was normal in every respect except for the hernia. There was no intolerance to exercise, no pain, and no change in color during or after exercising. The child has one younger brother who is well.

Physical examination revealed an obese, well-developed, alert, and active child. The patient appeared overly kinetic and fidgety to the point of being uncooperative. He seemed to be unduly conscious of his defect and constantly insisted on having it covered. His blood pressure was 112/80, pluse 90, and his weight 97 pounds. The red blood count was 4.61 million with 13 Gm. of hemoglobin. Urinalysis, serology, and Mantoux were negative. Except for the heart findings and the visible epigastric hernia no other abnormality was noted. There was a 3 by 12 cm. defect in the anterior abdominal wall extending from the xiphoid process down to the midabdomen. There was no visible umbilicus. Protruding through this defect and in the upper part of the hernia was a pulsating mass which expanded and contracted synchronously with the pulse. The pulsating mass which protruded considerably while straining could be grasped with the fingers. Firm squeezing did not interrupt the pulsations but did produce an occasional extrasystole. A Grade III systolic murmur was heard best just above the pulsating mass where a thrill was readily palpable.

Read at the sixth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 8, 1952.

The electrocardiogram showed an incomplete left bundle branch block and other minor abnormalities compatible with congenital heart disease.

Operation (Feb. 22, 1952).—A hockey stick incision was made from the left nipple to the xiphoid process and extended downward in the midline to below the level of the defect in the abdominal wall. Immediately beneath the skin the diverticulum came into view. The left pleural cavity was entered through the fifth interspace. The fifth and sixth ribs were cut at their junction with the sternum, and the xiphoid process was removed to insure adequate exposure. After the diverticulum was completely exposed a needle was inserted into its lumen and 20 c.c. of Diodrast was injected rapidly. Immediately after the injection a roentgenogram was taken which showed direct communication between the diverticulum and the left ventricle (Fig. 2). As the diverticulum was further isolated it was noted that the pericardial sac was missing on the anterior surface. Posteriorly there was a pericardial sac which, when opened, was found to be continuous with the thoracic portion. This posterior prolongation of the sac was fused to each side and to the blunt tip of the diverticulum. The pericardial sac was detached from the lateral and inferior surfaces of the diverticulum (see Fig. 3, B). The diverticulum had not come through the diaphragm but had pushed its way beneath the sternum and forced both leaves of the diaphragm downward without involving or distorting the pleural cavity.



A. *B.*

Fig. 1, *A* and *B*.—Photographs reproduced from a movie film. *A* shows the diverticulum in diastole, *B* shows it in systole.

The anterior surface of the heart was fixed to the posterior surface of the sternum making it impossible to reduce the cardiac portion of the hernia into the chest cavity. It was apparent that to correct the abnormality it would be necessary to amputate the diverticulum. A right-angled multitoothed clamp was placed across the neck of the diverticulum and slowly closed. In spite of complete occlusion the musculature of the diverticulum continued to contract for some time. The clamp was kept in place for five minutes. During this time a continuous electrocardiographic tracing revealed no change except that the bundle branch block was partially corrected. There was no change in the rate or character of the pulse nor in the blood pressure. The clamp was then removed to see what would happen. The diverticulum immediately filled and began contracting as before. No change in blood or pulse pressure followed but the electrocardiogram showed a recurrence of the bundle branch block. The clamp was reapplied at the base of the diverticulum, completely occluding its lumen, and it was amputated leaving a sufficient stump distal to the clamp to allow for adequate suture. The myocardium was approximated by means of several

interrupted mattress sutures. The endocardium was closed with a continuous suture of 00000 silk. A third layer of sutures was used to approximate the myocardium over the closed layer of endocardium (Fig. 3). There was no bleeding when the clamp was removed. The strip of pericardial sac, previously fashioned, was drawn over the stump of the diverticulum and sutured to the lower end of the sternum. The pleural cavity was closed in layers with continuous surgical gut. The fascial layers of the abdominal wall were dissected free and utilized to repair the ventral hernia. The child left the operating room in good condition.

The postoperative course was entirely uneventful. During the first forty-eight hours the pulse rate was rather rapid but no signs of pending decompensation were detected, and on the third postoperative day the pulse became normal. A roentgenogram of the chest taken on the tenth postoperative day revealed normal heart and lungs. There was slight elevation of the left leaf of the diaphragm. The child was discharged on the thirteenth postoperative day.

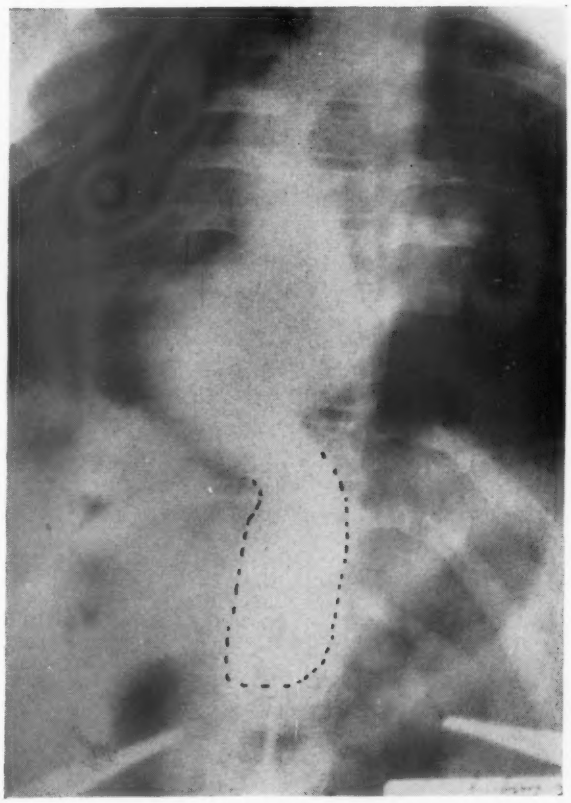


Fig. 2.—After the diverticulum had been exposed, 20 c.c. of Diodrast was rapidly injected and a roentgenogram made. The diverticulum is outlined.

Since discharge from the hospital the boy has been entirely well. A recent electrocardiogram still shows the minor abnormalities but all evidence of the bundle branch block has disappeared. The mother has noticed a marked change in the boy's personality; he is more affectionate and cooperative, fights less with his younger brother, and takes interest in activities which he previously avoided.

Pathology.—The specimen consists of a diverticulum of the heart weighing 42 grams and measuring 5 by 5 by 3.5 cm. (Fig. 4, *A* and *B*). The wall is of uniform thickness measuring 1.5 cm. The lumen of the diverticulum measures 1.5 cm. in diameter. The microscopic examination reveals normal epicardium, myocardium, and endocardium.

DISCUSSION

Etiology.—True diverticula of the heart may be classified as (*a*) traumatic, (*b*) degenerative, and (*c*) congenital. It is not too uncommon following injury to the myocardium to have an aneurysm or diverticulum form. Likewise, it is

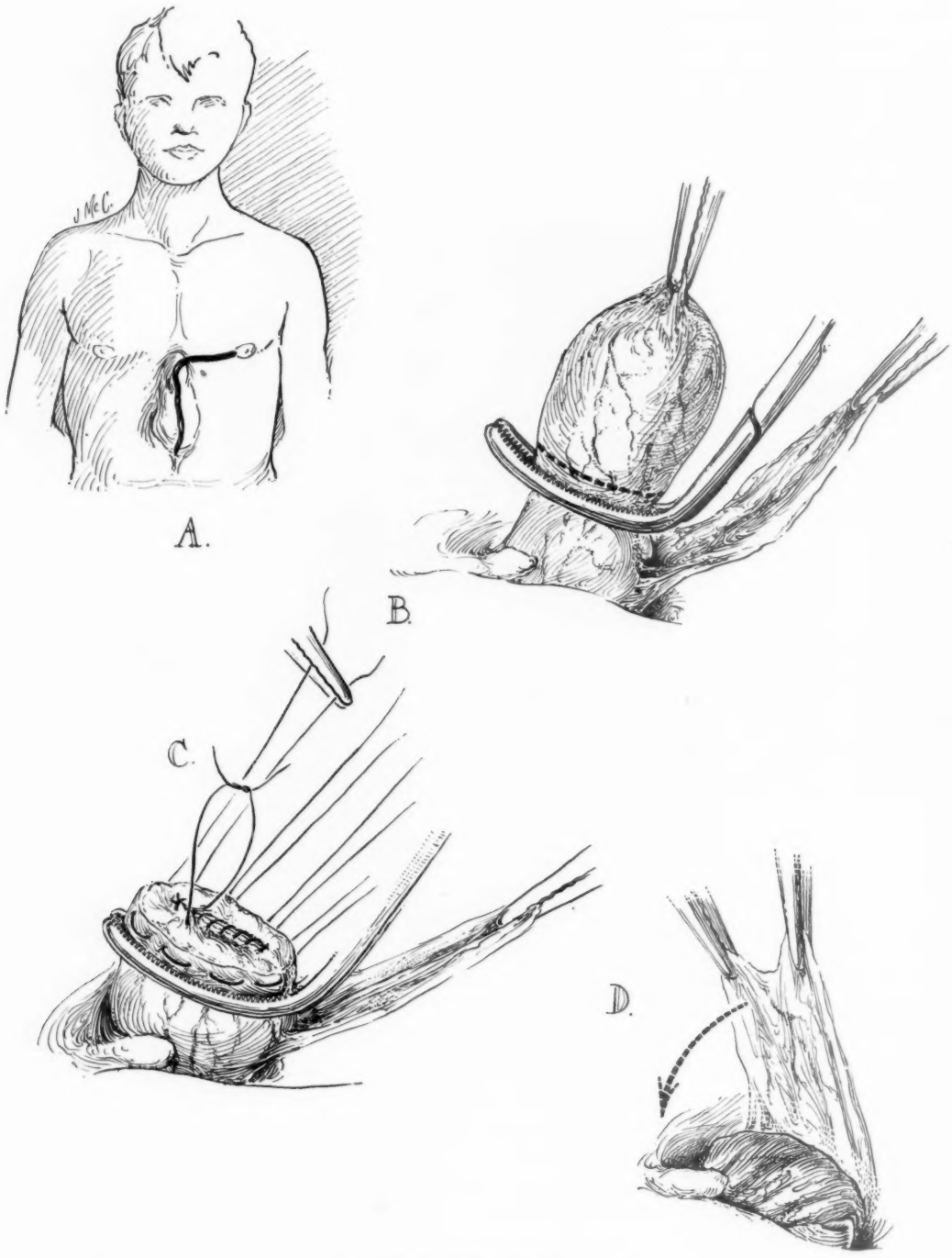
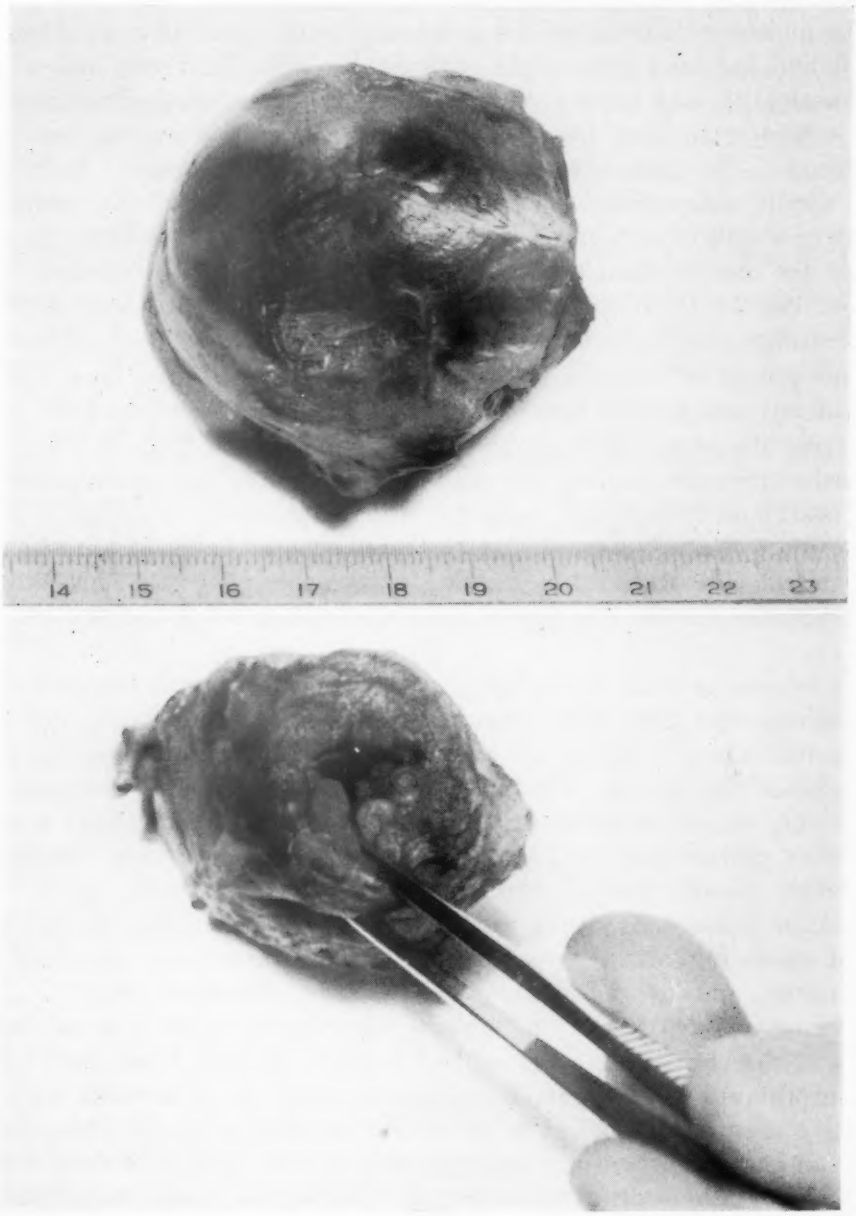


Fig. 3.—(See text.)

fairly common to see at necropsy a ballooned-out ventricle or an aneurysm at the site of a recent myocardial infarct. It is very rare, however, to see a true congenital cardiac diverticulum (see *Embryology*).

A.



B.

Fig. 4, *A* and *B*.—The diverticulum weighed 42 grams. The opening was 1.5 cm. in diameter.

Diagnosis.—A diagnosis of a congenital diverticulum is usually easily made. The association of a ventral hernia and a visible pulsating mass is almost conclusive evidence. However, the condition must be differentiated from ectopia

cordis, an anomaly which may also present a pulsating mass in the epigastrium. Of the 141 cases of ectopia cordis reviewed by Byron[5] in 1948, eighty-one were of the thoracic or pectoral type and thirty-eight were of the abdominal type. Ectopia cordis thoracica is easily recognized because of the characteristic defect in the thoracic cage and the wide fissure or cleft of the sternum. The abdominal type of ectopia cordis, although not as common as the thoracic type, is compatible with life and has been reported in patients who have lived well into adult life. This abnormality may appear very similar to that of a congenital diverticulum of the ventricle in that they both present a visible pulsating mass in the epigastrium. The pathology is markedly different however. In a case of ectopia cordis abdominalis the entire heart lies outside of the thoracic cage whereas in a case of congenital diverticulum the heart lies within the thoracic cage but the diverticulum protrudes as a pulsating mass. Diagnosis of either condition can be established by the use of roentgenograms and angiocardiographic studies which will demonstrate the presence or absence of a diverticulum.

Embryology.—The embryology of cardiac diverticula has been profoundly discussed but conclusions have been only conjectural. It has been proposed that during the early fetal period, in which the cardiac loop is being formed, intracardiac pressure on a structurally weak point may produce a diverticulum. Swyer postulates the etiologic basis for true diverticula as a defect in muscular attachment to the valvular ring at the point where the right anterior portion of the mitral ring fuses with the ring of the aortic valve. This explanation fails to account for the etiology of those diverticula not found at the valvular ring.

We believe, in order to explain the embryology of a true congenital cardiac diverticulum, that associated abnormalities must be considered, and that the diverticulum is only a part of the anomaly. As in any other congenital anomaly, the condition has degrees of severity which may vary. The abnormality then may be one, all, or combinations of several of the following: (*a*) a defect in the basilar pericardium, (*b*) a diverticulum of the ventricle located in the epigastrium usually ventral and cephalic to the diaphragm, as in the case reported, or associated with a ventral defect of the diaphragm, (*c*) a diverticulum consisting of all three cardiac layers, epicardium, myocardium, and endocardium, and (*d*) a defect in the anterior abdominal wall.

The ventral portion of the septum transversum gives rise to the basilar portion of the pericardium, the ventral leaf of the diaphragm, and, by fibrous tissue proliferation, the ventral cephalic part of the abdominal wall.[8, 9] An anomaly consisting of a defect in the basilar pericardium, a defect in the ventral portion of the diaphragm and an epigastric hernia must arise from some basic abnormality of the septum transversum. During the formation of the cardiac loop the point of greatest convexity, which Bremer[10] points out as the future left ventricle, comes in close proximity to the ventral part of the septum transversum. It is at this point that the epimyocardium of the cardiac loop fuses with the mesenchyme of the septum transversum and as the latter descends the attached epimyocardium is pulled ventrally and caudally. The endomyocardium follows the line of least resistance as in sinusoidal formation, and the

result is a true congenital diverticulum of the ventricle. Thus, with fusion of the epimyocardium of the cardiac loop to the ventral leaf of the septum transversum, a diverticulum of the ventricle, a defect in the pericardium, and a ventral hernia result. The presence of a defect in the diaphragm will depend upon the amount of the septum transversum involved in the process of fusion.

Treatment.—In view of the fact that rarely does a child with a congenital diverticulum of the ventricle survive infancy, we believe this abnormality should be treated surgically as soon as possible. The common fate of all the patients reported not treated surgically was death by spontaneous rupture of the diverticulum. The treatment of choice is amputation of the diverticulum.

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PROSTATIC CANCER

II. Regional (Pudendal Plexus) Block Anesthesia for Open Perineal Surgical Biopsy of the Prostate Gland

Perry B. Hudson, M.D.,* and Alex L. Finkle, M.D.,** New York, N. Y.

(From the Division of Urology, the Francis Delafield Hospital, and the Institute of Cancer Research, Columbia University College of Physicians and Surgeons)

THE usefulness of surgical biopsy of the prostate gland in detecting curable adenocarcinoma is receiving wider application today than at any other time since the advent of safe, perineal prostatic surgery. This development brings to the operating room an increasing number of patients who are above 50 years of age, including a considerable proportion who may undergo radical prostatectomy as a result of microscopic diagnosis of previously unsuspected prostatic cancer.[1]

Among aging males, the cardiovascular, pulmonary, and other degenerative diseases are found in high percentage. These disorders increase the anesthetic as well as the surgical risk.

Inhalation, intravenous, caudal, and spinal anesthesia have all been used for perineal surgery of the prostate gland. Each method has disadvantages in the poor-risk patient. Frozen section diagnosis of perineally biopsied prostate glands necessarily prolongs the operative period. Many patients suffering prostatic tumor withstand poorly long periods of either inhalation or intravenous anesthesia. Caudal anesthesia is nearly ideal, yet it carries a constant (15 to 25 per cent) failure rate and is difficult to give as a continuous anesthetic while the patient is in the exaggerated lithotomy position required for surgical exposure of the posterior lobe of the prostate gland. Spinal anesthesia has recognized disadvantages, although it is useful for short-duration procedures.

At any rate, it is apparent that there is a place for regional block anesthesia in perineal prostatic surgery. No long period of training in anesthesiology is necessary in order to effect adequate pudendal block anesthesia, in contradistinction to the considerable experience in anesthesiology required to administer satisfactory caudal anesthesia.

The male perineum receives nerve supply from the pudendal plexus, derived mainly from the third sacral segment of the cord (L_5, S_1 to S_4). The *rami musculares* of this plexus supply the levator ani muscles, an area of considerable interest in perineal surgical approaches to the posterior aspect of the prostate gland. The *rami visceralis* supply, among other structures, the bladder neck. The *n. pudendus* gives off the inferior hemorrhoidal branches which innervate the external anal sphincter and the anal skin; these branches, anastomosing with those of the *n. perinei*—also arising from the *n. pudendus*—supply the soft tissues through which the surgical dissection must pass.

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*Assistant Professor of Urology and Damon Runyon Senior Clinical Research Fellow, Columbia University College of Physicians and Surgeons.

**Resident in Urology, Francis Delafield Hospital.

Fig. 1.

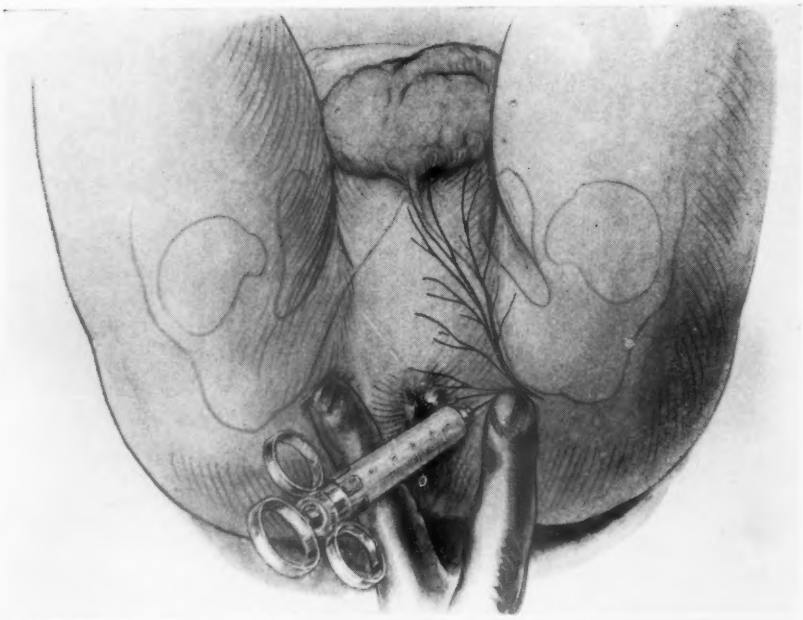

Fig. 2.

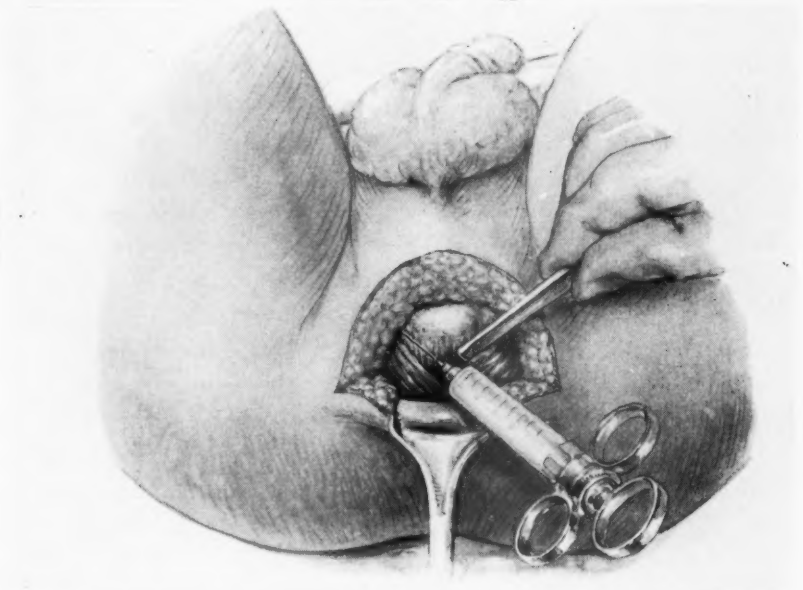

Fig. 1.—The ischial tuberosities are located by digital palpation, as illustrated. Ten to 15 c.c. of anesthetic solution is injected on each side. The main mass of pudendal plexus branches are effectively blocked by this injection alone. Bilateral injection is necessary.

Fig. 2.—Additional infiltration of anesthetic solution between levator ani musculature (bilaterally) and posterior lay of Denonvilliers' fascia to permit painless depression of rectum off base of prostate. Scalpel handle shown pushing aside levator ani on left. Needle tip located between levator ani and glistening posterior layer of Denonvilliers' fascia. Longitudinal fibers of rectum seen between needle and scalpel handle.

All of these nerves may be blocked by bilateral injection of the plexus at a point just medial to the ischial tuberosity (Figs. 1 and 2).

The additional injections toward the scrotum are made in order to include any branches from the *n. cutanei femoris posterioris* which may give overlapping sensory supply to the region.

PREMEDICATION

The following premedication schedule has proved satisfactory for pudendal block. It must be emphasized that any such schedule must be modified and adapted to the needs of the individual patient.

Night before operation:	Chloral hydrate, 0.65 Gm., orally, in solution.
Day of operation:	6:00 A.M. Repeat oral chloral hydrate, 0.65 Gm.
	7:00 A.M. Repeat oral chloral hydrate, 0.65 Gm.
	One-half hour preoperatively, Demerol, 100 mg. and atropine sulfate (or scopolamine) 0.0003 Gm., intramuscularly.
During operation:	For extreme restlessness or discomfort due to position, etc., Demerol or morphine may be given intravenously at the discretion of the anesthetist.

Further refinements in premedication and in perineal anesthesia are being currently investigated in this clinic and will be reported in a later communication.

CLINICAL EXPERIENCE AND RESULTS

With the method outlined, a total of twenty-two perineal prostatic biopsies have been performed under regional block anesthesia. The anesthetic agent was 1 per cent procaine solution in which 500 units of Alidase* was dissolved. About 50 c.c. of procaine was used as the solvent for the hyaluronidase,[2] but only 30 to 40 c.c. of the final solution was needed to accomplish anesthesia adequate for surgical maneuvers in the perineum. Before it became apparent that the second injections of procaine (Fig. 2) were necessary, there were four instances in which the analgesia was incomplete. Since the adoption of the present technique, no additional supplementary general anesthesia has been required in the latter eighteen cases, although some patients have complained of discomfort, which, nonetheless, appeared tolerable. Of interest was amnesia concerning the procedure in response to questioning the next day in most cases.

DISCUSSION

Although techniques for pudendal block anesthesia have been in use for many years, it has not often been utilized by urologists. The method described here is then, essentially, an application of an old technique for a new problem.[3]

In the course of a year's time it becomes desirable, on many urologic services, to perform biopsy of prostate glands for the suspicion of cancer only. When such biopsies are negative for malignancy, no further surgery is indicated. Under such circumstances, the least dangerous and least incapacitating

*Manufactured by G. D. Searle & Co., Skokii, Ill.

anesthetic is preferred. It has proved feasible to have hardy patients walk from their wards to the operating suite and, following prostatic biopsy under pudendal block anesthesia, walk back to their beds.

Within the framework of some clinical research projects on prostatic cancer there is found a necessity for serial prostatic biopsies. These biopsies are more easily justified and appear more readily acceptable to the patient if the anesthetic risk and discomfort are minimized. Local blocks have proved of great value in this regard.

In addition, the use of pudendal block does not interfere with anesthesia of another sort for transurethral or open prostatectomy by any surgical route. Such definitive procedures, when indicated, may be performed immediately after the frozen section diagnosis is reported. In this way, the delay caused by biopsy and frozen section does not lengthen the total period of anesthesia.

SUMMARY

1. The value of local (pudendal plexus) block anesthesia for open perineal surgical biopsy of the prostate gland in cancer work is cited.

2. The innervation of structures involved in perineal prostatic dissections is reviewed.

3. Premedication and anesthetic techniques are described and illustrated.

4. Satisfactory clinical results are mentioned.

5. The situations in which this anesthetic method is desirable are discussed.

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Case Report

STRANGULATED TRANSMESENTERIC HERNIA IN AN ERYTHROBLASTOTIC NEWBORN

(Report of a Case Treated by Resection and Anastomosis With Survival)

Sam C. May, M.D., and E. S. Brintnall, M.D., Iowa City, Iowa

(From the Department of Surgery, State University of Iowa College of Medicine)

TRANSMESENTERIC hernia is an uncommon lesion, only eighty-six cases having been reported with the majority of these occurring in adults. This case is that of the youngest patient yet reported with gangrene of involved bowel treated successfully by resection. The coincidental occurrence of erythroblastosis fetalis in this case is of interest.

CASE REPORT

C. M. (No. 52-3875), a 1-day-old white female infant, was admitted to the Pediatrics Service at University Hospitals on April 15, 1952, because of jaundice since birth. The mother had had four previous pregnancies and was said to be Rh negative. She had received a blood transfusion in connection with her last pregnancy. The patient was born of a normal delivery following a four-hour labor. At birth, jaundice was noted and the infant was referred to the University Hospitals for treatment.

Physical examination on admission revealed a jaundiced white infant weighing 3,100 grams. The rectal temperature was 98° F., the skin and sclerae were icteric. The abdomen was moderately distended aand peristalsis was hyperactive.

The Coombs' test was positive. The red blood cell count was 2,750,000 per cubic millimeter, the white blood cell count was 10,000 per cubic millimeter, and the hemoglobin was 9.7 Gm. per 100 c.c. Urinalysis gave normal results save for a one plus albumin.

On the day of admission, the infant was given a 150 ml. transfusion of Rh negative whole blood. The next day the abdomen was more distended and vomiting occurred. The rectal temperature was 101° F. On the third day of life, the abdomen was more distended, vomiting continued and dehydration became apparent. Physical examination at this time revealed the abdomen to be distended, tense, and quiet on auscultation. The abdominal skin was noted to be warm and red. The rectal temperature was 103° F. and the white blood cell count 10,500. Respirations were rapid and shallow. A roentgenogram of the abdomen revealed gas and fluid levels in dilated loops of proximal jejunum. A diagnosis was made of intestinal obstruction on the basis of a congenital anomaly. Perforation and peritonitis were suspected. Abdominal exploration seemed indicated despite the patient's grave general condition. Operation was performed April 17, 1952, under local anesthesia of 1 per cent procaine supplemented with nitrous oxide, oxygen, and ether. Upon opening the abdominal cavity, gangrenous small bowel was encountered and there was evidence of generalized peritonitis. Approximately 40 cm. of distal jejunum had prolapsed through a 2 by 1 cm. oval defect in the small bowel mesentery which was located in the region of the junction of ileum and jejunum. The prolapsed gangrenous bowel was resected, a two-layer end-to-end anastomosis was performed. The mesenteric defect was closed.

The postoperative course was uncomplicated. Fluids by mouth were well tolerated by the fourth postoperative day. The infant was treated on the Pediatric Service following her recovery from operation. Four 100 ml. Rh negative whole blood transfusions were administered to correct the anemia which was secondary to erythroblastosis fetalis. The pa-

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tient was discharged from the hospital forty-one days after operation, weighing 4,612 grams and taking her formula well. A letter from her local physician one month later stated that she was well weighed 5,200 grams, and was having normal stools.

DISCUSSION

The first reported case of transmesenteric hernia was by Marsh[1] in 1888. Since then numerous isolated cases and several collected series have been reported.[2-5] Dolton,[6] in 1943, was able to find seventy-six cases in the literature and added one of his own. We have found nine additional cases since then to give a total of eighty-six.

There are several theories as to the cause of this condition. One which has gained considerable acceptance is that of Treves[7] who described what is known as Treves' Field, an area in the mesentery of the terminal ileum between the last ileal artery and the ileocolic artery. Treves thought that a mesenteric defect was likely to develop secondarily in this area of congenital weakness. The majority of mesenteric defects occur in this region. Other possible causes of mesenteric defects are *(a)* primary developmental anomalies, *(b)* blunt abdominal trauma, and *(c)* postoperative mesenteric openings.[3, 8-10] In this present case it would appear that the defect was congenital in nature. Mitchell,[11] in 1899, observed a mesenteric defect in the ileocecal region in a newborn at autopsy. Long,[12] in 1927, reported a case in a newborn in which the abdomen was explored and nothing done because of the gangrenous bowel. At autopsy multiple fenestrations of the mesentery of the ileum were found. Bowel had herniated through one of these openings.

The condition appears with equal frequency in men and women and is predominately encountered in adult life. It has been stated[13, 14] that transmesenteric hernias account for from 8 to 11 per cent of all internal hernias. Of 335 cases of acute intestinal obstruction reported by McIver,[15] 0.9 per cent were due to internal hernias.

There are no specific signs or symptoms associated with transmesenteric hernia. The preoperative diagnosis has never been made. Patients are usually subjected to abdominal exploration with a preoperative diagnosis of acute intestinal obstruction or acute appendicitis.

Treatment consists of exploration with reduction of the hernia, resection of gangrenous bowel, and closure of the mesenteric defect.

The mortality rate for transmesenteric hernia with strangulation has been high. Cutler,[5] in 1925, reported a general mortality rate of 48 per cent with an 80 per cent mortality rate in those patients undergoing resection. More recent studies reveal a decrease in operative mortality. Cutler,[4] in 1944, and Johnson,[3] in 1950, reported mortality rates of 35 per cent in those patients treated by resection and anastomosis. Few figures are available concerning the results in infants. However, Glover[16] stated that the mortality from extrinsic obstruction in the newborn is from 25 to 37 per cent. The youngest patient treated by resection with survival was a 21-month-old child reported by Cutler.[4] In this instance Mikulicz's resection, rather than primary anastomsis, was performed.

SUMMARY

1. A case of strangulated transmesenteric hernia in a newborn infant with erythroblastosis fetalis is presented.

2. This is believed to represent the youngest patient to survive resection and primary anastomosis.

3. The possible causes, the clinical manifestations, and the treatment of this lesion are discussed.

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Editorial

Postoperative Care

THE most scientific of postoperative care cannot make amends for the complications of slipshod or unscientific surgery, hence for optimum effectiveness postoperative care must be preceded not only by a meticulous technique of surgery but also, and just as important, by the careful observation of sound anatomic and physiologic principles. The abuse of such basic concepts of good surgery as careful hemostasis or gentle tissue manipulation cannot be neutralized by transfusions or antibiotics.

Of all the numerous factors which contribute to a successful outcome following a surgical procedure, there is none more important than careful observation. Intelligent postoperative care does not lend itself to remote control, and someone familiar with the great variety of signs and symptoms diagnostic of changes within the surgical patient's economy must pay attention and interpret these meaningful messages in the light of possible prophylactic or therapeutic action. The sudden pallor, restlessness, air hunger, and rapid pulse, indicative of massive internal hemorrhage, are bits of evidence noted and interpreted by observation at the bedside.

There is a growing tendency on the part of surgeons to forego the correlation of laboratory tests with bedside patient evaluation and direct the postoperative care merely by the recordings in the laboratory report forms. Obviously such a policy exposes the physician to numerous dangerous therapeutic pitfalls and results in inadequate and erroneous observation of complications.

Of significance to the patient's general comfort and also to the ultimate recovery are many minor items, any of which alone might not seem important but in the aggregate contribute substantially to the patient's welfare and eventual recovery. Careful, intelligent, and constant observation alone can detect such signs. The examining finger, for example, is of greater value than paregoric in the diagnosis and therapy of a diarrhea resulting from a rectal fecal impaction. Subumbilical pain and discomfort accompanied by a suprapubic fullness following a spinal anesthetic or abdominal operative procedure may be relieved in most cases much more effectively by a catheter than by sedation. Intelligent laboratory tests can be carried out only by close patient-physician relationship as interpreted by frequent and careful inspection, palpation and auscultation.

A detailed, sequential recording of postoperative observations cannot be overemphasized. The more complicated cases involving problems of metabolism and electrolyte and fluid balance should be followed with composite charts, preferably attached to the patient's bed, permitting constant and thorough evaluation of the situation.

There is much lip service rendered the various salutary implications of mental comfort as related to the stress of surgery and yet, unfortunately, few doctors actually give this important part of postoperative care the serious consideration which it is due. For instance, the hushed sounds, strange smells, and bizarre sights of the hospital, which are an everyday part of the surgeon's life, may well be frightening experiences even to the bravest of patients, especially when goaded by the psychic trauma of the unknown. The doctor with little added effort and time, on occasion, may remove a patient from the throes of anguish and fear into the tranquillity of mental ease, and, parenthetically, those who do not pay attention to this phase of postoperative care are guilty of professional negligence.

The most frequently asked questions by those about to undergo surgery are related to the problem of pain, and the sympathetic surgeon in a conscientious effort to solve this situation may unintentionally jeopardize the patient's well-being, first, by the administration of analgesics prior to the determination of the exact cause of the pain, and second, by the lack of appreciation of the fact that postoperative discomfort is not always due to pain. Beecher,* in a study of 225 severely wounded soldiers in a forward combat area, found that three-fourths of the men requested no relief from pain. Under such circumstances small doses of a barbiturate plus small doses of a narcotic accomplished what large doses of either of the above alone failed to do.

An extremely significant phase of postoperative care is early ambulation which, in order to be effective, must be a systematic and planned program. One must never forget that the fundamental purpose of getting the patient on his or her feet is to initiate early muscle function with its beneficial side effects on vascular flow and various visceral metabolic activities. In the fervor of enthusiasm for early ambulation one should pause occasionally and take inventory to make sure that the plans undertaken actually are working out to the patient's benefit. Most of us have observed the spectacle of a staggering, pale, postoperative patient practically carried by a fluttering retinue of nurses, shuffling along a hospital corridor in complete exhaustion, giving scant heed to the encouraging verbal supervision of the surgeon; and this, all done in the name of intelligent medical care: How much more might have been accomplished by supervised exercises carried out in bed until the patient had gained sufficient strength for ambulation! Taking postoperative patients out of bed only to prop them up in chairs cannot be misconstrued as ambulation; in fact, such a practice, especially if the front edge of the chair causes pressure against the posterior aspect of the thigh, may actually be harmful and conducive to venous stagnation and thrombosis.

The systematic planning of a program of ambulation must commence on the admission of the patient to the hospital with simple detailed instructions not only on the technique but also on the reasons for such activities. Do not underestimate the intelligence of patients, for the simple physiology of vascular flow is readily appreciated by a people who live amid the complicated machinery of

*Beecher, H. K.: Pain in Men Wounded in Battle, Ann. Surg., 123: 96, 1946.

modern civilization, and if they are persuaded as to the reasons for such procedures then directions are carried out with meticulous care. The patient not only is instructed preoperatively on the benefits of actual ambulation, but also practices on the routine of bed calisthenics. Elman* describes a patient who was so thoroughly indoctrinated in bed exercises before surgery that while still under the influence of a general anesthetic following an abdominal operation he commenced going through the routine of specific calisthenic procedures lying on his side in bed.

Two factors related to the period of actual surgery are of importance in early ambulation and these are: first, the utilization of transverse or oblique, rather than vertical, incisions in abdominal surgery and second, the positioning of the patient on the operating table. Abdominal incisions which follow the major transverse and oblique muscle planes are not prone to cause as much postoperative pain, and hence the patient is less likely to fear early ambulation and more likely to give wholehearted support to the program. With the advanced techniques in anesthesiology and surgery which permit extensive operative procedures, both as to time and scope, it is imperative that the patient's position on the operating table be given careful consideration. In my estimation, every would-be surgeon should be adjusted on an operating table for a major procedure, including blood pressure cuff, intravenous fluids, and the other accessories of the operation, and forced to lie there for the necessary two or three hours without moving. Certainly if this additional surgical training were the rule, more care would be given the possibility of venous stagnation and nerve injury during the operation.

In résumé, postoperative care for optimum effectiveness must be preceded not only by a meticulous technique of surgery, but also, of equal importance, by the conscientious attention to sound anatomic and physiologic principles. Paramount among the numerous factors contributing to a successful outcome following any surgical procedure is careful, intelligent observation. The correlation of laboratory tests with bedside patient evaluation stands out as a vital component of intelligent observation, and a detailed, sequential recording of postoperative findings must be stressed for a thorough evaluation. Multifaceted indications for mental comfort are apparent in numerous phases of postoperative care. Emphasis is placed on the judicious consideration of analgesics, barbiturates, and narcotics. Of marked significance is the systematic planning for a program of early ambulation. There is a discussion of the relation of early ambulation to types of incisions in abdominal surgery and positioning of the patient on the operating table.

In an ultimate analogy the thoughtful person finds that the essence of real success in the outcome following a surgical procedure could be the essence of success in any other field—an underlying talent or drive, infinite care for details reaching a perceptive quality, long-range thinking, and a courageous imagination.

—*Melvin A. Casberg*

*Elman, Robert, *Surgical Care*, New York, 1951, Appleton-Century-Crofts, Inc.

Book Reviews

Fluid Balance; A Clinical Manual. By Carl A. Moyer, Professor of Surgery, Washington University School of Medicine, St. Louis, Mo., Pp. 191, with 1 illustration and 20 tables. Chicago, Ill., The Year Book Publishers, Inc.

Dr. Moyer's manual fulfills a need which long has been recognized by surgeons and other practitioners who seek a book on fluid and electrolyte balance which is applicable to everyday clinical situations. It is by a man who has contributed extensively to the basic understanding of this field and who, in addition, has acquired a wealth of experience in the management of problems of fluid therapy as they present themselves to the surgeon.

The early chapters are devoted to brief review of the fundamentals concerning the distribution of body water, the chemical composition of the fluid compartments, and the factors which underline alterations in fluid distribution and composition. A critical exposition follows the available clinical and laboratory criteria of fluid imbalance. Considerable emphasis is placed on the limitations of chemical examination of the blood and urine and on the importance of clinical signs in the evaluation of disturbances. A rather detailed outline of the physical signs associated with specific disorders of water and solute metabolism is perhaps the most valuable contribution which the book contains. The later chapters cover the use of repair solutions in the treatment of fluid imbalance, the complications of electrolyte therapy, and the problems which are peculiar to general surgery, neurosurgery, and urology.

This is a clearly written and useful book. It should be particularly well received by the great number of surgeons who tend to be frightened by the apparent complexity of this rapidly advancing field.

Psychosurgical Problems. Edited by Fred A. Mettler, M.D., Ph.D., Sc.D., (h.c.) The Columbia-Greystone Associates. Pp. 357, with 57 tables. Philadelphia, 1952, The Blakiston Company. $7.00.

This book represents the combined effort of the second group of investigators from Columbia University's College of Physicians and Surgeons and the New Jersey State Hospital at Greystone Park to evaluate the various procedures used in psychosurgery. In the main it is similar to the first report, although, in addition to topectomy, the surgical procedures include transorbital lobotomy, venous ligation, thermocoagulation of the cerebral cortex, and surgical thalamotomy.

This book includes chapters on the hospital management and social evaluation of the patients, the surgical procedures performed, an investigation of the patients from metabolic and neurologic aspects, and an evaluation of their psychologic status. In the last chapter the present status of the patients investigated and reported by the first group of the Columbia Greystone Associates is given. This is a two-year follow-up study which enhances the value of both books.

The emphasis of this latest report seems not to be on the conclusions reached, but rather on the methods of securing and evaluating data. These methods are not original, although they are relatively new and of interest. This book is of unquestioned value to anyone doing a similar type of research. It also should prove of interest to anyone working with the problems of caring for patients with psychogenic abnormalities.

Grundriss der traumatischen peripheren Nervenschädigungen. By professor Doctor G. Bodechtel, Düsseldorf, Doctor K. Krautzun, Recklinghausen, and Doctor F. Kazmeier, Düsseldorf. Pp. 106, with 78 illustrations. Stuttgart, 1951, Georg Thieme Verlag. D.M. 10.20. Agent for U.S.A.: Grune & Stratton, Inc., New York, N. Y.

This is a general discussion of nerve injuries without original or new material. It discusses the causes of nerve injury, general diagnostic features, and, briefly, regeneration and therapy.

The injection of Thorotrast to differentiate by roentgenography between total section and intraneural scar is suggested, but does not appeal to one who has seen fibrous tumor masses formed where Thorotrast has been injected into tissues.

Upper extremity nerve lesions are described in detail and constitute more than one-third of the book. Cranial nerve lesions and lesions of the lower extremity are more briefly discussed. Under "Sciatic Nerve Lesions" is included a short discussion of lumbar intervertebral disc lesions. Finally, there are sections on differential diagnosis, regeneration, and therapy.

For one who has need for a general treatise on nerve injuries in the German language, this book should be very satisfactory.

The Abnormal Pneumoencephalogram. By Leo M. Davidoff, M.D., Director of Neurological Surgery, Beth Israel Hospital, New York City, and Clinical Professor of Neurosurgery, New York University Postgraduate Medical School, and Bernard S. Epstein, M.D., Associate Radiologist, The Jewish Hospital, Brooklyn, N. Y., and Instructor in Clinical Radiology, Long Island College of Medicine. Pp. 506, with 695 illustrations on 289 figures. Philadelphia, 1950, Lea & Febiger. $15.00.

This book is the natural sequence of *The Normal Encephalogram* written by Davidoff and Dyke in 1937.

Part One concerns tumors of the brain and comprises approximately three-fourths of the book. Part Two concerns various nontumorous lesions of the brain, but tumors of the septum pellucidum are included here with defects and cysts of the septum pellucidum. The general form followed in discussing each group of tumors or each lesion is: a short introduction, discussion of the literature, the authors' material, summary, and differential diagnosis.

The introduction is a brief description of the tumor or other lesion, usually a well-selected short discussion of the clinical features and pathologic anatomy. The review of literature is quite extensive, resulting in sixteen pages of bibliography. Plain roentgenograms of the skull as well as pneumoencephalography are discussed both under literature and also under the authors' material.

The authors' material is based upon a review of all their pneumoencephalographic studies over a twenty-year period. The total number is not given, but it includes more than 500 cases of brain tumors.

For the most part, tumors are grouped for discussion according to location, such as frontal lobe tumors, lateral ventrical tumors, etc., rather than as tumors of a certain type. However, some tumors, such as olfactory groove meningiomas, craniopharyngiomas, acoustic nerve tumors, and others, are specifically discussed.

Many illustrative cases are reported in more or less detail.

The book is extensively illustrated, mostly with reproductions of roentgenograms, most of them greatly reduced and in some cases to the extent that the relevant features referred to in the text are not visible in the reproduction. Laminographic studies are a routine part of the investigation in cerebellar tumors and other midline tumors (p. 273).

There is obviously an error in the legend to Fig. 180.

This book should be most valuable to the radiologist, the neurosurgeon, and the neurologist as a reference in the interpretation of pneumoencephalograms.

Atlas of Genitourinary Surgery. By Philip R. Roen. Pp. 321, with 71 illustrations. New York, 1951, Appleton-Century-Crofts, Inc.

The author asserts that the purpose of this book is to teach, with the aid of simple line drawings, the common procedures of urologic surgery to the urologic resident, to the urologist who is confronted by an unusual surgical situation and wants to refresh his memory, and to the general surgeon who performs urologic operations infrequently. The book is arranged in nine sections dealing with the general principles of urologic surgery, and with operations upon the kidney, the adrenal, the ureter, the bladder, the prostate, the scrotum and testicle, the urethra, and the penis. The section on the adrenal is fragmentary and that on transurethral resection is inadequate. Considering the aim of the work, the descriptions of perirenal air insufflation and pneumoperitoneum could well have been left out in order to make the presentation of transurethral resection and adrenalectomy more complete.

The drawings are simple but clear and informative, and the book will undoubtedly be of great value to the groups named, and particularly to the beginning resident in urology, although he should certainly familiarize himself with a great many operations which are not mentioned in this book. It seems to me that the value of the work would be greatly increased by the inclusion at the end of each section of a few references in which could be found more details of the operations described, as well as descriptions of other procedures necessarily omitted from a book with the purpose of this one. The "Atlas" is well worth having.

Erratum

On page 969 of the December, 1952, issue, in Table II of the article "Clinical Studies on Effect of Barium in the Peritoneal Cavity Following Rupture of the Colon" by Norman Zheutlin, M.D., Elliott C. Lasser, M.D., and Leo G. Rigler, M.D., Minneapolis, Minn., the column headings "Died" and "Lived" should be reversed.

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teaspoon dosage
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